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MEDICINE

Pathologic Physiology of the Lung**

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It is an interesting observation that whereas most text-books of medicine will discuss fully the altered physiology that takes place in diseases of the various organs of the body, the pathologic physiology that occurs in pulmonary diseases is practically ignored. Although no explanation can be given for this, the result can only be confusion in the student's mind. Again, the present tendency to emphasize the importance of radiographic diagnosis, rather than its use as an aid to the final diagnosis in conjunction with the physical findings, has led to a regrettable trend of perfunctory examination of the chest.

This paper will attempt to review some of the altered physiological processes that take place in diseases of the lung. Only a few of the disorders of physics will be dealt with and the abnormal physical findings that result will be explained.

I

One of the most important features of the anatomical structure of the pulmonary system is its extraordinary richness in elastic tissue. This extends from the trachea throughout the finest ramifications of the bronchial tree to the alveoli. Again, in the mediastinum, intimately surrounding the various structures that fill that space and binding them together, is areolar tissue which also possesses elastic qualities. Lastly, surrounding the bronchial tree and acting as a support for the accompanying blood and lymph vessels, is the connective tissue. This structure, as well as the vessels it contains, also possesses elastic qualities, in that these elongate and shorten with the branches of the bronchial tree they accompany during the respiratory phases.

Mechanically, when an external force which is acting on an elastic solid body is released, this body, if perfectly elastic, will return to its initial size and shape. Similarly if this body is expanded, its potential elastic recoil increases in proportion to the extent of the force exerted upon it. This elastic recoil, represents its force of retraction and is equal to the force which has caused its extension. It other words, the forces of extension and recoil are equal and opposite, so that their algebraic sum will equal zero. These principles apply to the lung which is in reality an elastic body.

The lungs, which are really composed of one-third tissue, and two-thirds air, are, in the living being, in a constant state of tension, the degree varying with the inspiratory and expiratory phases. It is this tension with the consequent constant potential elastic recoil that accounts for the sub-atmospheric or "negative" pressure in the intrapleural cavity.

In the emphysematous lung, there is almost complete loss of elasticity and the intrapleural pressure fluctuates around that of the atmosphere. This leads to an increase in the functional residual air and expiration has to be performed by an active muscular effort. With the exception of the internal intercostal and anterior scalene muscles, this can only be performed by the accessory muscles of respiration, such as the pectorals. The diaphragm is a muscle of inspiration and, in the normal individual, is elevated during expiration by the elastic recoil of the lung. In emphysema, not only is the elastic recoil abolished but positive intrathoracic pressure is generated, which further impedes complete relaxation of the diaphragm during expiration. The inspiratory efficiency or "stroke" of the diaphragm is consequently diminished. The prolongation of expiration, which is a text-book sign of emphysema, is due to the active muscular effort in compressing the chest wall. The paradoxical movements of the soft structures of the chest, such as the supraclavicular hollows and the lower intercostal spaces, which are sucked in during inspiration, can be explained by the positive intrathoracic pressure.

In spontaneous mediastinal emphysema the mechanism is different. It has been shown that any sudden increased intra-alveolar pressure makes minute ruptures in the alveolar walls, especially if the elastic tissue is congenitally defective. If this pressure is great enough, air will pass through these ruptures and enter the perivascular sheaths of the finer branches of the pulmonary vessels. Further increase in pressure results in coalescence of the air bubbles to form larger ones and these gradually move medially towards the root of the lung through artificial channels produced in the vascular sheaths. At the hilum, these bubbles merge to form large blebs. With still greater pressure these break through into the mediastinal space producing a mediastinal emphysema. However, the air in the perivascular sheath may move peripherally towards the pleura where it forms a subpleural bleb; this generally

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occurring at the root of the lung. Rupture of this bleb produces a spontaneous pneumothorax. This helps to explain the reason for the interval that frequently exists between the actual act of exertion and the development of the pneumothorax:

II

In respiration, inspiratory enlargement of the chest is brought about by an active muscular elevation of the ribs and a contraction of the diaphragm; whereas, normally, the expiratory act is a purely passive affair. Respiratory movement of the chest is effected by two distinct and differing mechanisms. The upper part of both lungs, that is, the right upper and middle lobes and the left upper lobe and lingula, is expanded by a movement which is different from that which expands both lower lobes.

The muscles taking part in the active phase of inspiration are the scalenes and the external intercostal muscles. The former arise from the transverse processes of the cervical vertebrae and are inserted in the first rib, whereas the external intercostal muscles lie in the interspaces attached to adjoining ribs, running in an oblique direction downwards and forwards.

At the initiation of the act of inspiration, the scalenes contract and this moves the first rib upwards, fixing it in a more horizontal position. As a result, the manubrium, to which the first rib is attached, is pushed upwards and forwards. This results in an increase in the antero-posterior diameter of the upper part of the thorax and causes a slight expansion of the anterior part of the apex of the lung.

The next phase is performed by the adjoining five ribs, that is, the second to the sixth. All these ribs are distinguished by the fact that their articulating ends with the vertebrae run in a horizontal direction. Consequently, when the external intercostal muscles contract, the ribs will be pulled upwards owing to the fixity of the first rib. Because of the transverse vertebral articulation, the anterior end of the rib will move forwards and upwards with only slight lateral movement, resulting in a displacement of the sternum in that direction. This causes an expansion of the upper lobes, which will necessarily be mainly in a forward direction with little movement laterally. There will thus be very little sliding of the visceral and parietal pleurae over one another in this portion of the chest.

The lower lobes of the lungs are expanded by the movement of the next four ribs, that is, the seventh to the tenth. The remaining two ribs or "floating ribs" are inserted into the abdominal muscles and do not take part in the inspiratory act. These four ribs are characterized by the fact that their ends articulating with the vertebrae do so in a very sloping manner. Consequently, on elevation of these ribs, the movement will be entirely lateral in a "bucket-handle" fashion, with resultant widening of the sternocostal junction. Taking into consideration rib-action alone, expansion of the upper chest by the forward movement of the sternum is equal to the expansion of the lower chest by the lateral movement of the ribs. However, the latter movement also works in conjunction with the diaphragms, which contracts on inspiration and moves downwards like a piston, thereby expanding the lower lobes in a vertical direction.

In ordinary quiet respiration, the expiratory phase is largely a passive affair. The scalenes and the external intercostal muscle relax and allow the ribs and sternum to return to their original position by gravity. The abdominal muscles, which had relaxed in order to accommodate the displaced viscera caused by the descent of the diaphragm, now contracts. However, in forced expiration the internal intercostal muscles, which lie medial to the external ones and run in an opposite direction, namely, downwards and backwards, will by contraction cause a further apposition of the ribs. The abdominal muscles also actively contract still further, thus increasing the intra-abdominal pressure and aiding the ascent of the diaphragm.

From the above remarks, it will be understood that the lung does not expand like a balloon in all directions uniformly, but that the upper lobes expand chiefly in the antero-posterior diameter, whereas the lower lobes expand laterally and downwards. The greatest expanse will take place in the periphery of the lung, the least occurring naturally at the hilum. The two layers of the pleurae will slide over each other at their greatest extent in the region of the lower lobes explaining the reason for the occurrence of pleural rubs in this area. There are also portions of the lung where scarcely any movement takes place at all. The reason for this will be readily understood when it is seen that these regions are the posterior medial part of the superior portion of the chest as well as the costo-vertebro-mediastinal area. Thus a considerable portion of the lung affected by the poor expansion will be the apex, much of the upper lobe, and the upper part of the lower lobe. Expansion of this underprivileged portion of the lung can only take place by the inspiratory descent of the diaphragm which causes a pull on the hilum in a downward and forward direction.

This point is of great importance because it explains the predeliction of tuberculous lesions and cavities for this super-radicular portion of the lung. Tubercle bacilli tend to lodge and create tubercles where the movements of air and lymph-flow are least. So, if the movement of the hilum of the lung is interfered with, the expansion of that portion of the lung will also be restricted and the develop

ment of tuberculous disease will be greatly facilitated. There are so many causes of inflammatory enlargement of the hilar lymph glands, which result in increased connective tissue formation and anchoring, that this may explain the preferential localization of tuberculous superinfections in the apex, whereas the primary tuberculous lesion may be found anywhere and more often in the lower lobes.

It is thus evident that movement of the thoracic cage is a basic act in respiration, and it has been shown that the upper and lower parts of the chest move in different directions. As the underlying lung expands, the trachea and the bronchi with the attending connective tissue and vessels will elongate during inspiration and contract during expiration. The bronchi will also actively expand during inspiration and the opposite happens on expiration. It will be understood from this that if a portion of the underlying lung is diseased, whether in the parenchyma or bronchial tree, or if there is a foreign substance in the pleural cavity which compresses the underlying lung, there will be limitation of expansion of that portion of the lung. Expansion of the diseased area will progress only to the extent possible by the normal tissue which may still be present. Similarly, although the ribs normally move together in unison, ribs overlying a diseased portion of the lung will show diminished movement whereas the remaining ribs will move normally.

It is these finer delayed movements that will be missed by the usual method employed in the physical examination of the chest. By laying the palms of hands with the fingers upwards, only the forward movement can be appreciated, and is of use only in the upper anterior chest. What is more important, however, are the lateral movements of the chest especially in the lower parts. These movements are best assessed by gripping as much of the chest wall as possible in the palms and fingers and compressing it by bringing both hands together so that the thumbs touch in the mid-line. By watching the thumbs as the patient inspires, slight diminution or delay in expansion can be more easily detected. Alteration in movement implies disease, either in the underlying pleural cavity or lung. It is one of the earliest manifestations of disease, and is by far the most important sign in the physical examination of the chest. Even in bilateral disease, extent of disease is rarely equal and one side will move less than the other.

III

Breath sounds are produced by the inrush of air into the bronchial tree as the lung is stretched by the enlarging chest in inspiration. However, the sounds produced in the bronchial tree are of a different character than that which is heard by the listening ear over the chest wall. Although the sounds are damped down somewhat by passage through the chest wall itself, this is of minor importance.

The lung possesses a remarkable acoustic quality in the fact that it acts as a selective transmitter. It possesses the faculty of being able to transmit efficiently a relatively narrow band of frequencies, while tending to suppress or damp all other vibrations. The optimum frequency for vibration through normal lung parenchyma is around 250 to 400 cycles per second, which is a comparatively low-pitched note. It so happens that the sound produced in the bronchial tree is predominantly of a much higher pitch, ranging between 1,000 to 2,000 cycles per second. When this high frequency sound radiates through the bronchial tree and reaches the lung parenchyma, the selective transmitter action of this tissue fairly effectively suppresses these higher notes, allowing only the low frequency band to pass through. This results in a low-pitched note, the vesicular breath sound, which is normally heard over the whole lung.

Just as sound is produced in a labial organ pipe, the actual breath sound itself is produced by the eddies caused by the column of air striking the angles of the bifurcating bronchi. Air entering the alveoli causes a sudden stretching of their walls and the resultant vibration produces its own added sound as well. In expiration, the column of air strikes the bronchial wall directly opposite the orifice, again producing eddies in the current.

The lung can be compared to a sponge in which all the air spaces are lined by an oil film. The selective transmission of low frequency vibrations of the parenchyma is most likely due to the reflection of sound as it strikes the air surface in contact with the alveolar wall or the oil film lining the air-space in the sponge. This is similar to the effect of a pleural effusion or pneumothorax. Although both air and fluid are excellent transmitters of sound, the breath sounds are diminished or absent in these conditions. This is due to the reflection which takes place as the vibrations strike the pulmonary surface of the air or fluid in the pleural cavity and again when the vibrations strike the surface in contact with the thoracic wall. Similarly, percussion which sets the air-containing parenchyma in vibration, will produce a low-pitched note over normal lung tissue, because of the selective action again.

As may be readily surmised, alterations in the physical state of the lung parenchyma affect its sound transmission characteristics as well as its sound production ability. As a result there is a

profound alteration in the audible breath sounds in the particular area affected by the changes. With the appearance of some process which tends to disturb the normal structure of the lung parenchyma, as for example, a marked increase in the interstitial tissue or inflammatory exudate in the alveoli, there is a corresponding impairment of the efficiency of the lung parenchyma as a selective transmitter. As a result more of the higher frequency notes are permitted to pass through to the chest wall, the pitch of the breath sounds becoming raised and broncho-vesicular breathing is heard. With an increase in this replacement process, there is a proportionate increase in the higher frequency notes that are heard in the chest wall. When this process has reached its maximum degree and the normal lung parenchyma has become completely solidified, then its ability to act as a sound producer and selective transmitter is completely nullified. It now becomes an excellent transmitter of all sound frequencies and the sound heard is "bronchial breathing." Similar results will take place in the percussion note and a dull to flat note will be produced depending on the degree of underlying disease.

IV

The protective mechanism of the bronchial tree is performed by the ciliated epithelium which is present as far as the respiratory bronchioles, the cilia waving in an undulatory fashion carrying mucous or foreign particles upwards toward the trachea. The normal parenchyma of the lung, the mucosa of the bronchi and at least the lower part of the trachea are practically sterile. The ciliated epithelium plays a predominant part in this sterilization, aided by the bactericidal qualities of mucus and phagocytosis.

The second line of defense in protecting the bronchial tree is the cough, which is a reflex induced by irritation of the tracheobronchial tree or laryngeal mucosa. It consists of two actions, deep inspiration followed by a violent expiratory blast. Two phases should be distinguished in the expiratory part. In the first, the glottis remains closed while the expiratory muscles contract and raise the pressure within the lung. This is called the "compressive" phase. In the second, the glottis is slightly opened while expiration still goes on, so that a violent draught of air is produced, tending to expel any material in the bronchus.

In the smaller bronchi and the peripheral lung parenchyma, where the air blast is feeble or impossible, the substance requiring expulsion is continuously wafted upwards by ciliary action, and by the "tussive squeeze" is forced upwards to the larger bronchi where expulsion is completed by the expiratory blast. When local disease or injury has destroyed the cilia, the powerful tussive squeeze, alone, is ordinarily adequate. This tussive squ occurs at each cough by the powerful thor muscles compressing the invaded lobe, forcing the secretion in a way comparable to manual c pression of a rubber sprinkling bulb.

The natural methods of emptying the trach bronchial tree are extremely inefficient. In upright position, secretions from the upper resp tory tract are continuously draining into it passing the glottic barrier, and even in the sup position, lipiodol will enter the bronchi w placed in the hypopharynx while the patien heavily asleep, all without inducing the co reflex. Again, when lipiodol is inserted int bronchopleural fistula, the cough reflex is elicited until the larger bronchi are reached.

By virtue of the upright position of the b the accumulation of mucous exudate will be m marked in the more dependent bronchi and expulsion will be hindered by gravity. The r common bronchus and its division are m vertical than the left and their diameter is latively larger. Therefore, the expulsion of mu is more difficult from the lower bronchi, especia those on the right. This will explain why obstr tion of these bronchi will be more easily realiz and why lower lobes are more frequently involv than the upper and the right more than the l Purulent material lying stagnant in the bron is acted on by saprophytic organisms. T changes the character of the material by alter the viscosity of the exudate and making it eas to be expelled by the tussive squeeze. The e pulsive force of the cough is considerably ha pered by the contraction of the lumen and short ing of the bronchi which occur during expirati and even more so, on coughing. The cough a scatters pus throughout both lungs and the d inspiration, which precedes the cough, drives pus more distally.

V

That the portion of the bronchial tree drain a diseased area in the parenchyma is alw affected, is an undoubted fact. The extent of involvement will naturally vary depending on extent and nature of the disease. The patency the draining bronchus to the affected area play very important role in the progression of pathological process.

The same factors apply in dealing with p monary cavities, tuberculous or otherwise. Th excavations in the lung result from a necrosis the lung tissue with evacuation of the necr material through the draining bronchus, wh although diseased, must necessarily be pate otherwise a cavity could not occur. The spheri shape of the cavity is due simply to the differe in pressure inside and outside the cavity. In

interior of the cavity, the pressure is equal to that of one atmosphere, provided, of course, that the cavity communicates freely with the outside world. Around the exterior of the cavity, the pressure is equal to the intrapleural pressure, which is subatmospheric, ranging normally about -7 mms. Hg., but is often more negative, owing to atelectasis and fibrotic shrinkage of the surrounding lung tissue. Thus, the external pressure of the cavity being lower than the internal one, the cavity is compelled, for obvious mechanical reasons, to assume a spherical shape.

The evolution of the cavity will depend on the fate of the draining bronchus. If the bronchus remains patent, the cavity will preserve its size and shape. If it becomes obstructed, the air in the cavity will become absorbed by the same mechanism which takes place in atelectasis and the cavity will collapse. If a check-valve obstruction develops in the bronchus, allowing the free entry of air into the cavity but obstructing its exit, the cavity will increase in size, resulting in a tension type of cavity.

Atelectasis is a phenomenon which complicates any pulmonary disease, only when bronchial or bronchiolar obstruction is present, regardless of the nature, bacteriology or pathology of the disease which has produced it. However, in a number of pulmonary diseases atelectasis is of paramount importance in the development and evolution of the disease itself. Lobar pneumonia and bronchopneumonia have been shown to be, in the initial stages, really cases of atelectasis secondary to obstruction of a bronchus or smaller bronchioles. Tuberculosis and anaerobic infections appear to be especially influenced by atelectasis. This happens in the latter, because anaerobic organisms cannot develop and grow unless previous bronchial obstruction and atelectasis has deprived the pulmonary tissues of a considerable amount of oxygen contained in it. In tuberculosis, however, as the tubercle bacillus is an aerobic organism, the absorption of oxygen following atelectasis hampers and even stops further development of the tubercle bacillus, and by suppression of the circulation which occurs in atelectasis, leads to tissue anoxaemia and fibrosis.

These principles are made use of in the treatment of pulmonary tuberculosis. Rest in bed in the decubitus position will restrict movement in the already underprivileged portion of the lung, which, as has previously been pointed out, is the favourite site of lodgement of the tubercle bacillus and for tubercle formation. The restricted movement will favour kinking and blockage of the already diseased bronchus. In artificial pneumothorax the diminished movement of the affected lung due to the increased intrapleural pressure will further diminish the lymphatic and blood supply to the diseased atelectatic area leading to increased fibrosis. It will also be understood that cavity closure will take place in both artificial pneumothorax, thoracoplasty or any other form of collapse therapy, not by active compression of the cavity itself but by kinking the draining bronchus with the resultant absorption of air.

VI

The mechanism of air absorption is similar in an obstructed bronchus, pleural space, and any pulmonary cavity. Of primary importance is the circulating blood. In fact, if the pulmonary artery draining the affected lobe of the lung is ligated before its bronchus is blocked, atelectasis will not take place. The mechanism of air-absorption can be simply explained if an alveolus is taken as an example. This is an elastic air-containing sac separated from the perialveolar capillaries by the alveolar epithelium. This partition acts as a wet membrane in the diffusion of gases between the alveolus and the blood. The extent of the diffusion will depend on several factors, the most important being the partial pressures of each gas on either side of the wet membrane.

If a mixture of gases is contained within a closed space, it is not the percentage of each individual gas that is important, but that each exerts its own pressure as if it alone made up the whole mixture. Atmospheric air is composed of roughly 21% oxygen, 0.03% carbon dioxide, 79% nitrogen, with a variable amount of water vapour and traces of neon and argon. As the atmospheric pressure at sea level is 760 mm. Hg., the partial pressures of each gas will be as follows:

Oxygen = 21/100 x 760 = 159 mm. Hg.
Carbon dioxide = 0.03/100 x 760 = 0.2 mm. Hg.
Nitrogen = 79/100 x 760 = 600 mm. Hg.

In the alveolar sac, the composition of the air is slightly different owing to the gaseous exchanges which are constantly taking place. The percentage and partial pressures of the individual gases, which are saturated with water vapour, now become as follows:

Oxygen 15% (pressure 130 mm. Hg.).
Carbon dioxide 5% (pressure 40 mm. Hg.).
Nitrogen 80% (pressure 590 mm. Hg.).

Because of the gaseous exchanges which are constantly taking place in the tissues, the gaseous content of the venous blood is again different. These are as follows:

Oxygen 5% (40 mm. Hg.).
Carbon dioxide 6% (45 mm. Hg.).

Nitrogen is an indifferent gas, not being capable of entering into chemical combination with substances in the blood, and is only slightly soluble. Consequently there is very little difference between its content in the alveolar air and the blood.

This quality is a highly important one, however, in that it acts as a "brake" preventing the too rapid exchange of gases between the alveoli and the blood. Without this brake, complete atelectasis would occur in a matter of minutes instead of taking one to two days. Gaseous anaesthetics, not being affected by nitrogen in the same manner, are more rapidly absorbed and this will account for the rapid appearance of post-operative atelectasis.

The sequence of events can be more easily explained if they are visualized as a successive series of individual gaseous exchanges. In actual practice, however, it must be understood that all these gaseous exchanges are going on simultaneously.

As the alveolar air contains 15% oxygen in contrast to the venous oxygen which is 5%, ten volumes of oxygen will diffuse into the venous blood. Similarly, one volume of carbon dioxide will diffuse out of the venous blood, which contains 6%, into the alveolus with its 5%. The alveolus has now lost nine volumes of gas and the oxygen and carbon dioxide come into equilibrium in the venous blood and alveolus. However, the alveolus, being an elastic sac, accommodates itself to the new volume. This results in an increase of the percentage and pressure of nitrogen which now becomes 89% and 675 mm. Hg. respectively. As the venous content of nitrogen is 80% with a partial pressure of 608 mm. Hg., nine volumes of nitrogen will diffuse out of the alveolus into the venous blood.

The alveolus again contracts to accommodate itself to its new volume and this results in a relative increase in the percentage and partial pressure of oxygen and carbon dioxide. For purposes of explanation, suppose that oxygen alone is increased relatively. This causes a rise to 14% in the oxygen content, so nine volumes of oxygen will diffuse out of the alveolus into the venous blood. The carbon dioxide is now relatively increased by nine volumes to 15%. This increases the partial pressure of the carbon dioxide in the alveolus and nine volumes of carbon dioxide diffuses out. Thus the cycle continues until all the gases of the alveolar air are absorbed.

VII

Finally, a word must be said regarding clubbing of the fingers. Although the actual cause is still undetermined, its occurrence in almost any variety of pulmonary, pleural, or mediastinal disease makes it a most important aid in arriving at a clinical diagnosis. Although most common and pronounced in chronic suppurative lesions, it can also occur in any condition causing chronic pneumonitis, atelectasis, or external compression of the lung in chest deformities. It consists of an increased proliferation of all the tissues of the finger tip, but most apparent about the fibrous elastic tissue of the nail bed. There is increased thickness of the walls of the blood vessels as well as the formation of new capillaries, with an increased peripheral blood flow and a raised intra-arterial pressure within the digital arteries.

That a large factor in its causation is anoxaemia is undoubted. However, the anoxaemic theory does not explain its occurrence in infective states such as subacute bacterial endocarditis. But in these infectious processes, the sedimentation rate which is dependent on rouleaux formation, is increased. In fact, intravascular rouleaux have been observed in this condition and it will be evident from this that the difusion surface per unit of haemoglobin will be necessarily reduced. This together with the increased blood flow and blood pressure which will facilitate the circulation of the rouleaux, will result in tissue anoxia and finally clubbing.

Clubbing is first detected because of thickening of the fibro-elastic tissue of the nail-bed. Ordinarily, the plane of the proximal portion of the nail makes an angle of about fifteen degrees with the dorsal plane of the bone. This angle is best determined by having the finger flexed at the knuckle and viewing the terminal phalanx from the side. There are normally great variations in the sagittal curvature of the nail which is often mistaken for clubbing. This error will not be made if the normal angle between the plane of the nail root and the bone is observed. A decrease in this angle is the first sign of clubbing. If it becomes obliterated or negative the clubbing is usually manifest.

Laboratory Aid in the Diagnosis of Epidemic Neurotropic Virus Diseases

With Special Reference to the Collection of Specimens*

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Introduction

Of recent years there have been many outbreaks of epidemic nervous diseases due to viruses described in North America. In Manitoba, for example, there have been large outbreaks of encephalitis lethargica (von Economo), poliomyelitis, Western equine encephalomyelitis, and perhaps infections due to other viruses. In 1947, as in 1941, it is probable that poliomyelitis and Western equine encephalomyelitis were both prevalent simultaneously.

The clinical diagnosis between abortive and atypical forms of poliomyelitis and Western equine encephalomyelitis is notoriously difficult, especially when it is remembered that similar clinical pictures may be seen in infections due to many other viruses, such as those of herpes febrilis, mumps, St. Louis encephalitis, and lymphocytic choriomeningitis.

Of recent years, many advances have been made in the laboratory diagnosis of virus diseases of the nervous system. The methods available include the inoculation of pathological material in experimental animals and fertile eggs, and the testing of convalescent sera for antibodies. It is probable that in future the Provincial health officers can arrange for a strictly limited number of such tests to be performed on cases of epidemic disease in Manitoba, and requests should be addressed to them in the first instance.

Collection of Material

Nervous Tissue

When a case of nervous disease proves fatal, portions of the brain and spinal cord should be removed with aseptic precautions. Portions of the cerebral cortex, basal ganglia, brain stem, and cord are placed in 50% sterile glycerol in saline, and stored in an ordinary refrigerator until tests can be performed. A duplicate set of tissues should be placed in small screw-cap vials in an ice box packed with solid carbon dioxide, thus ensuring a temperature of about —60°C. Needless to say, a third set of material will be collected and examined histologically, using special stains to demonstrate nuclear inclusions.

*Portion of an address on "Recent Advances in Neurotropic Virus Diseases" delivered before the Winnipeg Medical Society on January 12, 1947.

Cerebro-Spinal Fluid

In non-fatal cases, especially where clinical evidence of meningeal irritation is present, it is always worth obtaining a sample of C.S.F. Full bacteriological, chemical, and cytological tests are first performed, and the remainder is then frozen at —60°C pending inoculation in animals and eggs.

Stools

In the diagnosis of poliomyelitis, three consecutive stools are obtained, and frozen at —60°C in individual containers, such as small "fruit sealers." Washings of naso-pharyngeal secretion may also be obtained, and similarly frozen.

Sera

It is always necessary to have at least two samples of serum, one obtained as early in the illness as possible, and the second 10-14 days after onset. As the antibody rise is sometimes slow (e.g. in lymphocytic choriomeningitis), a three-month sample should also be taken.

Serum should be separated from blood as soon after clotting as possible, and should then be frozen at —60°C, without any heat inactivation. Serum that has not been frozen is not altogether satisfactory for testing, as the virus neutralizing antibodies may decline in titre. At any rate, both samples must be treated by the same technique.

Examination of Material in Laboratory

Brain and cord tissue is ground with sterile abrasive in a mortar, and diluted with sterile saline or buffer to an approximate 1/10 suspension. The material is then lightly centrifuged, and the supernatant used as inoculum. It is probably desirable, if sufficient animals are available, to inoculate also weaker dilutions such as 1/100 and 1/1,000. Material is inoculated cerebrally in mice and guinea pigs. Rabbits may also be used. Eggs are widely used and inoculated by various routes. Inoculation tests should detect the presence of St. Louis, equine encephalomyelitis, mumps, lymphocytic choriomeningitis and some other viruses. Poliomyelitis virus can only be demonstrated by inoculation of monkeys.

Stools are inoculated in rhesus monkeys by the nasal route, using raw material. The stool is usually suspended 1/5 or 1/10 in distilled water, and the supernatant used after settling on the bench for a few minutes. Alternatively, stools or naso-pharyngeal washings are treated with ether and inoculated cerebrally, peritoneally, or intradermally in rhesus monkeys. As these animals are not uniformly susceptible, and as the amount of virus present may be small, it is recommended that three be inoculated with each sample, thus rendering the capital expenditure on monkeys alone about $75.00 to $100.00 per patient, apart from

feeding and maintenance costs, which may reach 50 cents to $1.00 per day per monkey. There is no other diagnostic test of value in poliomyelitis, except the inoculation of monkeys with brain, cord, stool, or nasopharyngeal washings.

Sera are tested by the **virus neutralization test** and by the **complement fixation technique.** Tests are readily available only for the following viruses: St. Louis, Western or Eastern equine encephalomyelitis, lymphocytic choriomeningitis, mumps, and influenza.

In the virus neutralization test, the dilution of virus that kills 50% of a series of mice is discovered. Virus-human serum mixtures are likewise titrated, and if the serum contains antibody, it will be found that mice can only be killed by a much stronger dilution of virus. Acute phase sera in an area where a disease is endemic may contain a small amount of antibody, but in convalescence a very considerable rise in antibody occurs.

The complement fixation test is carried out on the lines of the Wassermann reaction. The antigen consists of mouse brain infected with the virus; complement is the usual guinea-pig serum. Complement fixing antibodies are not usually present in acute phase sera, but in the presence of a neu tropic infection should be readily found in convalescent sample. Fixation tests are avail for **St. Louis, equine encephalitis, lymphoc choriomeningitis,** and **mumps.**

Conclusion

In conclusion, laboratory tests for neurotr virus diseases are accurate and reliable, but only be carried out in a laboratory especi equipped for the work; even so, no large num of tests can conveniently be handled. The num of animals involved in the diagnosis of even case may be very large, perhaps even upward 100, and the cost is proportionate. However sirable it may be to have facilities for the diagn of virus infections approaching in scope efficiency those that have been available for s years in the field of bacteriology, the techn nature of the work renders this goal quite attainable for many years. In the meantime, h ever, by careful selection of material, much be learned from the performance of comparati few tests, which can be undertaken in th laboratories in the Dominion already study virus problems.

GYNECOLOGY

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Vaginal Hysterectomy

Dr. W. J. Friesen

Although vaginal hysterectomy is not a new surgical procedure, it has enjoyed until recently only sporadic approval. While its acceptance is becoming more generalized, there still remain islands of strong resistance. Recent enthusiastic publications, by over emphasizing the technical simplicity of, and claiming rather extravagant uses for this operation, have aroused renewed condemnatory criticism. One can only conclude from these articles that the operation is ill-conceived, unnecessarily radical and more in the nature of a stunt than a serious effort in surgery. Those familiar with the fundamental principles of vaginal hysterectomy will hasten to disagree with this view. It is quite true that vaginal hysterectomy is a relatively simple operation, quick of performance and having very low morbidity and mortality rates. But these very advantages are partly responsible for its indiscriminate use. Gastro-enterostomy is at least one operation that has fallen into disrepute for similar reasons.

Vaginal hysterectomy has been performed for almost every disease that the uterus is heir to: Fibroids, cancer of the cervix and fundus, chronic inversion of the uterus, uterine bleeding from all causes in all ages and procidentia. It is markable that the proponents of the clamp meth frequently place procidentia last on their list indications, while those of the ligature meth place it first. This suggests a fundamental ference in both concept and technique betwe the two methods. The ligature method is practical in the nullipara, but in the repair birth trauma it would appear to be a super technique.

By the process of morcellation a uterus grea enlarged in size by fibroids can be removed by vaginal route. It is difficult to see where advantage lies in so doing when the abdomi approach provides much greater facility, is acco panied by much less bleeding and eliminates danger of overlooking other attendant pathol in the pelvis. There are, of course, cases in wh vaginal hysterectomy is the method of choice will be shown later. Nevertheless, a good rule follow is never to attempt the operation when uterus is larger than the foetal head at term.

Vaginal hysterectomy for the control of so-call intractable uterine haemorrhage is a pernici practice if it is not preceded by endometrial biop and, in the younger woman, adequate clini investigation and medical treatment. This appl

particularly to the adolescent who may recover spontaneously once a true menstrual cycle has become established. Rarely one encounters a case in which all conservative methods are of little avail, or in which protracted medical treatment is impracticable for social or economic reasons. However, the important decision to make is not one of approach but rather for the need of hysterectomy itself.

Insertion of radium is still the most favoured treatment for menopausal bleeding. But it is well to remember that the uterus at this age is a useless organ and harbours malignant potentialities. Further, it is becoming increasingly apparent that the incidence of corpus cancer is greater in women who have had severe menopausal bleeding and/or menopausal doses or radium. It follows, therefore, that the need for conservatism is not nearly so great in this group and that, when other conditions demanding it also exist, vaginal hysterectomy is the treatment of choice.

The results obtained in the treatment of cancer of the cervix with deep x-ray and radium have not yet been seriously challenged by surgery. In a few skilled hands abdominal hysterectomy has compared favourably. But the vaginal route is certainly impracticable because of the extensive dissection required to make surgical treatment complete. Nevertheless, vaginal hysterectomy offers tempting possibilities in the treatment of the very early case of cervical cancer. At present, however, there is no place for vaginal hysterectomy in the treatment of cancer of the cervix or corpus uteri.

In connection with procidentia, it is interesting to note that cancer of the cervix is of rare occurrence. This behaviour of the cervix would appear to challenge the importance of chronic irritation in the etiology of cancer because it is an invariable feature of procidentia[1].

The Le-Fort operation is frequently used in the treatment of procidentia in patients who are elderly or otherwise considered poor operative risks. While this procedure provides good support with a minimum of surgical trauma and operating time, it has certain objectionable disadvantages. It disregards normal anatomical relationships and is incomplete: the weight of the uterus and the diseased cervix are still present and vaginal drainage is sometimes seriously impeded. With average skill very little more time is required for vaginal hysterectomy which, with its admittedly low morbidity and mortality even in the aged, will provide the same degree of support without the above objections. Further, with the uterus removed one will never be called upon to contend with such conditions as cancer or pyometra in an organ left almost inaccessible by a previous surgical procedure.

Not uncommonly a vaginal repair and hysterectomy are indicated as in the patient with birth trauma and fibroids of the uterus. If the patient's condition permits, first one and then the other operation is done in one continuous process. But this often proves too shocking to the patient and the second part has to be postponed. If the uterus is not too large and freely movable, a vaginal hysterectomy should be done. This will eliminate the possible need for a second operation and is very little more shocking than either operation in itself.

Vaginal hysterectomy finds its greatest usefulness in the treatment of procidentia in the woman past childbearing age. It should be regarded, however, only as part of an operation that includes repair of the anterior and posterior vaginal walls. In birth trauma with prolapse of the uterus, the utero-sacral ligaments are necessarily stretched and the pouch of Douglas often deepened. Enterocoele of some degree is therefore usually associated with procidentia. The ever present diseased cervix and malignant potentialities of the uterus have already been mentioned. By doing a vaginal hysterectomy these elements of birth trauma are simultaneously and effectively removed.

To understand how this is brought about a few features of vaginal hysterectomy by the ligature method will be elaborated[2] although it is not intended to discuss technical details here. The posterior cul-de-sac is opened and the utero-sacral ligaments carefully identified and secured with strong clamps. Once the uterus has been removed, the cul-de-sac and recto-vaginal septum are explored for depth and enterocoele. If the pouch of Douglas is deep and enterocoele is present, this is dealt with by closing the peritoneum much as one does any hernial sac. The bladder is then freed from the underlying pubo-cervical fascia and the uterosacral ligaments attached to the latter near the anterior pubic rami on either side. The purse string suture used to close the peritoneum is tied around the utero-sacral ligaments to further reduce their slack and secure them in the midline. This procedure not only reduces the slack in the utero-sacral ligaments, but provides strong ligamentous continuity from the symphysis pubis in front to the sacrum behind. The repair of the vaginal walls and the perineum is then completed in the usual manner. When this procedure is properly executed recurring prolapse is very unlikely to occur and the depth of the vagina not appreciably decreased.

Summary

In the past as in the present, misguided but well intended enthusiasm has led to some grave abuses of vaginal hysterectomy. The indications for this, as for any other valuable surgical operation, should never be allowed to exceed its limitations. These are fairly definite: cancer of the corpus or cervix uteri, large fibroids, a fixed uterus due to any causes or the presence of other disease in the pelvis not necessarily affecting the uterus but requiring close inspection and possible surgical removal. In the patient past childbearing age the need for conservatism is lessened bec the useful life of the uterus has expired and possibility of cancer developing in it become increasing menace with succeeding years. Vag hysterectomy is, therefore, difficult to excel in treatment of procidentia in women of this group. It is an operation easily tolerated elderly patients and has none of the disadvanta of the Le-Fort operation.

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CANCER

Edited by D. W. Penner, M.D.

Delays in Cancer Diagnosis

D. W. Penner, M.D.*

It is well recognized that most cancers in their early stages can be cured. Applications of the modern skills of surgery and/or radiation make almost all early cancer accessible to treatment.

In 1938 Pack and Gallo published data on 1,000 cases which were analyzed to determine the responsibility for the delay in the treatment of cancer. They considered the patient at fault if he delayed more than three months after the first symptom to consult a physician, or if he refused to follow acceptable advice. The physician was considered at fault if he failed to arrive at a diagnosis in a month, or if unable to do so, he did not refer the patient to a suitable person, or if he failed to apply proper treatment. On this basis the patient alone was responsible in 44.3%, patient and physician 18.0%, physician alone 17%, with no delay in 20.7%. It is clearly shown that there is still room for improvement in the services rendered by the medical profession to patients suffering from cancer. It was also noted that failure on the part of the physicians to make early and correct diagnosis is not confined to the internal group of neoplasms but also included cancers of the breast, cervix, lower rectum and oral cavity.

In September, 1947, Leach and Robbins published a "follow-up" analysis to the above survey, based on 500 cases studied at Memorial Hospital, New York. When it is seen that there are an estimated 300,000 new cases of cancer in the United States (Canada 30,000), and that each year 150,000 people die of this disease (Canada 15,000), it is of greatest importance that the medical profession does not take the problem of early diagnosis lightly. In Manitoba in 1946 there were 1,356 new cases of cancer, 335 of which were first reported only after the patient was dead. In the same year 905 persons died of cancer. 75% of early can of the breast and 95% of early cancers of the can be cured whereas in advanced disease salvage rate may be less than 5%. Using the s criteria as Pack and Gallo, Leach and Robb found that the patient alone was responsible 32%, patient and physician 10.8%, physician a 27.8%, no delay 29.4%. They conclude, ". study . . . suggests strongly that the patient making progress in reducing the delay, altho the physician is not."

One hundred case histories taken from records of the Manitoba Cancer Institute w analyzed to determine the causes of delay in diagnosis of cancer. No deliberate attempt selection of cases was undertaken except t necessitated by the presence or absence of s ficient data. These one hundred cases inclu all types of neoplasms. On a basis of these hundred cases the average delay from the onse the first symptom until the patient sought med advice was 5.2 months, and the average time t elapsed prior to the establishment of correct di nosis and treatment by a physician was 3.9 mon The longest delay by a patient was 36 mon The longest delay on the part of a physician also 36 months. 28 patients presented themsel to a doctor within one month of the first sympt In 48 cases the doctor made a correct diagn and instituted adequate treatment within a mo of first seeing the patient. In only five cases the patient consult a doctor and the doctor in tute treatment within one month. Analysis of causes for delay on the part of the physic showed them to fall within three main grou wrong diagnosis and therefore wrong treatm failure to make a diagnosis in spite of a cert amount of investigation; and thirdly, failure recognize that an abnormal state existed.

The first group of wrong diagnosis inclu such things as injecting hemorrhoids in cases

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rectal carcinoma without doing a rectal examination, treating tumors as inflammatory lesions and carcinomas of the larynx with gargles. Failure to establish diagnosis only too often was due to lack of complete history and physical examination, and was not necessarily confined to obscure internal tumors. In several cases, patients, in spite of definite symptoms, were assured by their physician that the symptom was not abnormal and were told to forget about their complaints.

Using the same criteria for responsibility for delay as Pack and Gallo, the patient was responsible in 47% of the cases and the physician in 45% of the cases.

Comment

Nothing holds more promise for the immediate reduction of cancer deaths than reduction in the delays in diagnosis and treatment. In our own small series, as well as in other quoted series, the physician is only too often at fault in causing unnecessary delays in the diagnosis and proper treatment of patients.

Complete histories and physical examinations could prevent many of the unnecessary mistakes in diagnosis.

Further education of the medical profession as to the urgency of early diagnosis and treatment is needed.

Conclusion

The cure of early cases of cancer can only be accomplished if there is no delay in diagnosis and treatment. The medical profession can do much to accomplish this end.

I wish to thank Mrs. M. E. N. Macdonald, of the Manitoba Cancer Institute, for the assistance in securing the records on which much of this paper was based.

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Abstract

Occupational and Post-Traumatic Cancer. Bull. N.Y. Acad. Med. 23: 145-162 (March), 1947. Fred W. Stewart, New York.

The problems of occupational and post-traumatic cancer are thoroughly discussed.

Occupational Cancer: Certain cancers are clearly related to industry and these include tumors of the urinary system from exposure to betanaphthylamine, skin cancers in paraffin, shale oil, arsenic, tar and chimney sweeps. The fallacy of casual studies to demonstrate relationships is pointed out, e.g., it would be faulty to conclude because nasopharyngeal cancer is common in the Chinese and since many Chinese work in laundries that this tumor is related to the laundry industry.

Post-traumatic Cancer: Single uncomplicated trauma and its alleged relationship to production of various types of tumor is discussed.

Bone Sarcoma: Critical analysis of case histories leaves much doubt as to the acceptability of trauma in bone sarcomas. Evidence of a negative character is presented which in the author's opinion outweighs the supposedly direct evidence of a positive character. The alleged precipitating trauma is often mild and yet the author does not see bone sarcomas following the maximum trauma of fractures. Nor do we see tumors following extensive and repeated surgical trauma to bone. Longstanding osteomyelitis and the lesions of Charcot joints or syringomyelia do not lead to tumor.

Mammary Carcinoma: No convincing evidence has ever been put forward that trauma can cause mammary cancer. A factor often lost sight of is that the changes leading to the development of mammary cancer probably extend over a five-year period or more, and during this period of time a tumor cannot even be suspected, yet many of the alleged traumas exciting mammary cancer fall well within this period.

Tumors of the Testis: Tumors histologically similar to those arising in testis occur in the ovary where trauma seldom if ever can be a factor. Also the incidence of tumors in abdominal testis is higher than when they are in the scrotum. Developmental disturbances appear to be a factor in production of testicular tumor.

Several case histories are presented which allegedly proved a single blow to have produced a tumor. The author points out the numerous fallacies in these cases. These cases cannot stand up under even superficial criticism. Yet this faulty reasoning and lack of accurate reliable data are being used continually, especially in compensation cases, to prove that trauma can cause tumor. In order to prove relationship we should be able to fulfill the following—"Prior integrity of the part, exact extent of injury, specific tissues injured, known time factors." We have only one good source of material where all these factors can be studied and that is in surgical trauma, yet no one seriously believes that surgery produces cancer due to its trauma.

The role of trauma in accentuation of tumor: This question is not easily answered and much will depend on our increasing knowledge of the natural history of the tumor in question.

D. W. Penner.

CARDIOLOGY

Edited by J. M. McEachern, M.D.

Notes on Tachycardia

A. B. Houston, M.D., F.R.C.P. (C)

Tachycardia

Definition: Abnormally rapid heart rate—or, on the average a rate over 80-90 in Adults, 150 in Infants, 120 in Pre-adolescents.

A. Physiological Causes

(1) **Normal**—Rarely a rate above average normal may be normal for that particular individual. However, a rate above 100 in an adult in the resting state may be considered abnormal.

(2) **Normal reaction to various stimuli such as** Ingestion of food and what I call "Physical, Mental or Emotional Strain"; for example — exertion, excitement or tension, anxiety, etc. It must be remembered that the emotional strain may not be obvious—but other tension signs such as excess perspiration, cold, clammy hands, dilated pupils will usually be present. The general practitioner who knows the personality of his patient and the emotional stresses and strains to which he is subjected will be in a better position to spot an "Emotional Tachycardia" than the specialist. The Syndrome of Neuro-circulatory Asthenia (D.A.H. —Soldiers' Heart—Da Coste's Syndrome) probably falls in this group as recent Psychiatric studies of these cases have revealed. A sleeping pulse rate is generally normal.

(3) **Voluntary Tachycardia** — extremely rare — patient can, at will, increase heart rate—which increase is associated with other signs of Sympathetic N.S. activity.

B. Extrinsic Causes

i.e., tachycardia due to disorders other than heart disease

(1) **Reduced Venous Return**—as in shock or haemorrhage—also the tachycardia following prolonged standing is ascribed, by some, as due to decreased venous return. Such cases, however, usually exhibit greater or lesser degrees of Neuro-circulatory Asthenia and emotional causes are difficult to exclude here. There is said to be "pooling" of blood in periphery leading to decreased Venous Return, but this is open to considerable doubt. It is interesting to note that the Syndrome of Postural hypotension has been (I believe, wrongly) ascribed to a similar mechanism—yet in this syndrome there is absence of rise in pulse rate on assuming upright position (together with marked fall in Systolic and Diastolic B.P. and inability to sweat)—a fact which illustrates our lack of detailed knowledge in the subject.

(2) **"Toxic" Cases:** (a) Fever—usually each degree Fahrenheit rise causes 10/min. rise in heart rate.

(b) Thyrotoxicosis—a factor which must be considered in every case of unexplained rapid heart action—regular or otherwise. The most difficult differentiation to make is from Cardiac Neurosis with tachycardia (N.C.A., etc.)—and I have found that simply shaking hands with the patient (in a room temperature of 65-75° F.) is extremely helpful. The hands of the Thyrotoxic are warm and moist—those of the neurotic are cool and clammy.

(c) Drugs: (1) Via Sympathetic Stimulation—Adrenalin—caffeine—thyroid, etc. In my experience Tea, Coffee, Tobacco, are rarely primary etiological factors.

(2) Via Parasympathetic Paresis — Atrophine and related compounds cause Vagal paralysis (1/15-1/20 gr. = complete paralysis).

(3) Others—such as alcohol, histamine, etc. After an alcoholic binge tachycardia may persist for 24 hours or more.

(3) **Vagal Neuritis:** Theoretically any cause of peripheral neuritis may cause paralysis of Vagus and tachycardia—actually Diphtheria and Poliomyelitis are only two of any frequency. Cachexia may also be included.

(4) **Miscellaneous:** Pulmonary Embolism is major one to be mentioned here.

All of the causes mentioned to date result in a Sino-Auricular tachycardia—i.e. a tachycardia with a normal Physiological rhythm—due, in the last analysis to a Primary or reflex decrease in Vagal tone or increase in Sympathetic tone to the heart. Such tachycardias are regular and usually less than 150 per minute and require no treatment in themselves but treatment directed at removal of causative factor.

C. Intrinsic Causes

i.e. Tachycardia due to Cardiac Disease

With the exception of Pericarditis with effusion and chronic constricture pericarditis, where the mechanism of tachycardia appears to be via Bainbridge reflex as a result of decreased Venous return to heart, Tachycardia, at rest, in presence of Organic Heart Disease is associated with greater or lesser degree of heart failure either left or right sided or both. It is obviously impossible to discuss the causes of heart disease here—but may I make a plea for avoidance of Diagnosis "Organic Heart Disease" since in more than 90% of cases an etiological diagnosis as follows will be clinically possible:

(1) Congenital Heart Disease — long history often of aid.

(2) Rheumatic Heart Disease — associated, if tachycardia is resulting, with either obvious Valvular disease or an Acute Rheumatic episode.

(3) Luetic—Blood Serology will help—a regurgitant aortic murmur is luetic until proven otherwise.

(4) Thyrotoxic—other signs of this state will usually exist.

(5) Hypertensive and (or) Atherosclerotic disease—presence of the high blood pressure—other signs of atherosclerosis, a history of angina or coronary occlusion will help.

(6) Cardiac Tamponade — constrictive pericarditis or acute pericardial effusion—diagnosis by the classical physical signs.

(7) Rarer causes of Heart Disease—such as various types of myocarditis, Beri-Beri heart, etc.

Unless such a diagnosis can be reasonably and almost obviously made it is preferable to avoid mentioning heart disease to the patient at all—as, in general, cardiac disease is over, rather than under, diagnosed. This is especially so where murmurs are found and "leaky valves" are diagnosed. May I offer a tip or two on the assessment of cardiac murmurs—often a most difficult task.

(a) Systolic murmurs—it is not well enough realized that careful listening will reveal a systolic murmur in some 70% of normal people at one time or another. There are two points that will aid—(i) the murmur to have significance must be constantly present at all times and in all positions. (ii) On a basis of intensity systolic murmurs may be graded from 1 to 4—where grade 4 is one associated with a thrill; to be of significance a systolic murmur should be at least of grade 2 intensity. Even when very definite, systolic murmur rarely means serious disease—assessment is better made on other signs such as enlargement. The murmur simply suggests that we look for real evidence of cardiac disease. Further, basal systolic murmurs are seldom of significance.

(b) Diastolic murmurs — signify organic heart disease until proven otherwise which is rarely possible.

The treatment of the tachycardias in this group resolves itself into the prevention and treatment of heart failure which we cannot deal with here.

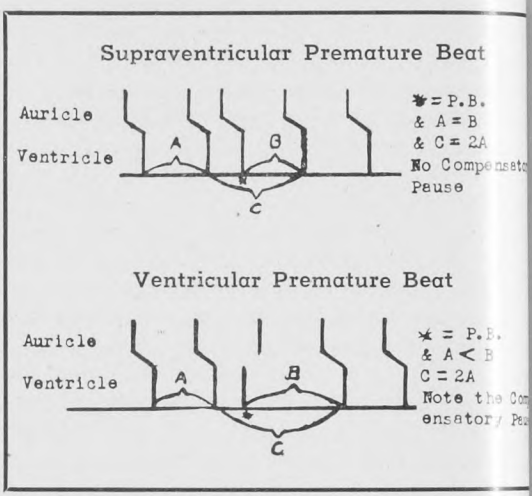
D. Abnormal Rhythms Associated With Tachycardia

I. Extra Systoles — (Premature beats — Ectopic Beats).

Incidence: "A very rare individual escapes having premature beats at some time of his life." —White.

Mechanism: An Impulse arises outside normal pacemaker (or rarely in the normal pacemaker at an abnormal time) usually before the next bea due, but always after absolute refractory perio the tissue in which it arises—and then spr throughout the ventricular muscle giving ris a mechanical systole.

Types: Many may be differentiated accor to the site of origin of the impulse but bro two types may be recognized—(a) Supraventric —in which the impulse arises in the A.V. n auricles or S.A. node. (b) Ventricular—in w the impulse arises in one of the branch bundle in the ventricular muscle itself. This type is t as common as the other.



Etiology: Sex incidence is about equal w increasing incidence with age. The majority cases occur in the absence of heart disease. Toba tea, coffee, alcohol, etc., are always mentioned etiological factors but their role is obscure. E tional disorders often seem to be a factor.

Symptoms: These are usually absent. If pr ent they seem to be related more to the comp satory pause than to anything else. "My he turned over," "Stops," etc. If extra-systoles p sist, in the absence of a nervous reaction to th symptoms disappear.

Diagnosis: It may be stated initially that p mature beats are not generally associated w tachycardia and, in fact, the usual variety dis pear with the tachycardia of exertion. The di nosis is made (a) by the E.K.G. (b) Clinicall there is usually a basic regular rhythm interrup by premature beats which in the common t are followed by a compensatory pause. It must recalled that the premature beat may be too we to reach the wrist, so that the diagnosis should made at the apex and, in fact, the only relia place to study irregularities is at the apex. illustration may clarify the diagnosis of ext systoles.

Multiple irregular extrasystoles may be impossible to differentiate from auricular fibrillation without recourse to special methods.

When Are Extra-Systoles of Significance?

(a) They are one manifestation of digitalis intoxication and may go on to pulsus bigeminus wherein there is an alternation of normal beat and Ventricular extra-systole—each extra-systole being followed by a compensatory pause; or Pulsus trigeminus i.e. normal beat, 2 Ventricular extra-systoles, compensatory pause and repeat, carried further, this may result in ventricular tachycardia —a dangerous rhythm.

(b) In Mitral Stenosis — extra-systoles when multiple may presage the onset of auricular fibrillation.

(c) In Myocardial Infarction — extra-systoles may presage onset of Ventricular Fibrillation.

Prognosis

Mackenzie, J.—1913. "When extra systole is the only . . . sign, the prognosis is a favorable one, and where it is associated with other signs the prognosis is to be based upon these other signs."

Treatment

(1) In the absence of symptoms—none—don't even mention them.

(2) With non-disabling symptoms reassurance only.

(3) With digitalis poisoning discontinue the digitalis.

(4) In the presence of other disease treat the other disease.

(5) In (c) above, treatment is indicated to prevent ventricular fibrillation. This is accomplished by the use of quinidine, and as much as two grams (30 grains) may be needed initially.

In (b) above, quinidine is indicated to prevent the onset of auricular fibrillation in dosage of 0.6-1 gram (10-15 grains) or more, daily in divided doses at four hourly intervals preferably on an empty stomach.

(6) Disabling symptoms without cardiac disease are usually attributed to a cardiac neurosis and may be treated as such; sedatives, quinidine (0.6-1 gram daily) and possibly potassium salts are the drugs to be used.

II. Paroxysmal Tachycardia

Definition: A recurring regular tachycardia of about 200 per minute, of sudden onset and offset, and variable duration and frequency, unrelated to organic heart disease although such may exist in less than 10% of cases, in which case the tachycardia imposes a serious strain on an already compromised myocardium.

Mechanism: A series of regular rapidly recurring extra-systoles.

Types: Supra-ventricular and ventricular. The supra-ventricular type accounts for over 90% of cases. The ventricular type occurs with usually serious organic heart disease and is thus of serious import. The diagnosis of the type can be made only by the E.K.G.

Etiology: Obscure—fatigue, exertion "indigestion," tobacco, alcohol, tea, coffee, allergy, are all mentioned but their significance is not clear. Deep seated emotional disorders also seem to be a factor. Heart disease occurs slightly more frequently in these cases than in normal individuals. In the majority of cases no cause is shown, although the apparently high incidence of this arrhythmia in Wolf-Parkinson-White syndrome should be noted.

Symptoms: Abrupt onset of rapid heart action with one or more of palpitation, choking sensation, faintness, rarely anginal pain and almost constantly anxiety, fear and apprehension.

Signs: Rapid regular heart action which is unchanged by exercise, position, respiration, etc.

Diagnosis: Made mainly on the history.

Prognosis: As in extra-systoles, is based on the associated disease, if any.

Treatment: 1. Of the Attack:

(a) Carotid Sinus pressure.

(b) Other vagal stimulation—by eyeball pressure, induced vomiting, a drink of cold water, forced flexion of trunk, etc. Many people discover for themselves how to stop the paroxysm.

Mecholyl may be used in stubborn cases in doses of 20-30 mgms.—but may induce vomiting, asthma, fall of blood pressure, heart block, etc., which may be relieved by atropine.

(c) Quinidine 6 gr. every two hours for five doses daily may be tried.

(d) Rarely, as a last resort in desperate cases, one of the purified Digitalis glucosides in doses of from 0.2 to 1.2 mgms. may be used intravenously; this is not recommended.

(e) Sedation—not morphine—may be used in conjunction with any other measure.

2. Prevention of Subsequent Attacks if Possible

(a) Elimination if possible of all fore-mentioned etiological factors.

(b) Marked strong repeated explanation and reassurance to not only help prevent attacks but to prevent subsequent cardiac neuroses.

(c) Quinidine may be used in daily dosage of about one gram as for extra-systoles.

(d) Digitalization is rarely effective.

(e) Potassium salts are used.

(f) Sedatives are of distinct value.

III. Auricular Flutter

Regular rapid (300+) auricular contractions with varying auriculo-ventricular (A.V.) block (2:1, 3:1, 4:1 usually) resulting in a regular rapid

ventricular rate which may be irregular if the degree of A.V. block varies from moment to moment.

Mechanism: Is said to be a regular circus movement in the auricles resulting in impulses reaching the A.V. Node at a more rapid rate than they can be accepted with a resultant functional A.V. block and ventricular rates of ½ - ⅓ - ¼, etc., of auricular rate.

Etiology: Usually organic heart disease—in 90% of cases. Especially mitral stenosis, hypertension, thyrotoxicosis and coronary heart disease.

Precipitating factors are sudden effort, excitement, trauma, and surgery (especially thoracic surgery). Sex incidence is 3:1 male—all ages are affected but 75% cases are over age 40.

Symptoms: In brief are those of the underlying disease plus consciousness of rapid heart action and symptoms similar to cases of paroxysmal tachycardia.

Signs: Usually a tachycardia which is usually regular.

Diagnosis: (a) By E. K. G.

(b) Clinically—if neck veins can be well seen a venous flutter at a rate of 200-400 may be compared with the apical rate and the diagnosis established.

(c) Carotid sinus pressure may cause a slowing of apical rate to ½ or less but this will be for the duration of the pressure only and such pressure does not abolish the abnormal rhythm as it does in paroxysmal tachycardia.

Prognosis: As to the duration of the attack is variable, but the response to treatment is better than in paroxysmal tachycardia—rarely the abnormal rhythm may last weeks, months or even years. As to life the prognosis is that of the underlying cardiac disease.

Treatment: 1. Of the Attack:

(a) Digitalis is the drug of choice if the attack lasts more than a few hours. Dose is about 9 grs. daily in divided doses for two-three days, then 1-1½ grains daily if the abnormal rhythm persists.

(b) Quinidine may be used following digitalis if normal rhythm does not occur.

(c) Digitalis usually either stops the flutter or induces fibrillation; if fibrillation occurs and the underlying cardiac disease is well compensated, Quinidine should be used in an attempt to restore normal rhythm. In presence of congestive failure, cardiac enlargement, or defects of conduction quinidine should probably not be used.

2. Prevention of Attacks: When, spontaneously or due to therapy, the initial attack subsides one should determine the underlying cardiac disease if any and treat it. Then:

(a) If flutter recurs in the absence of any heart disease it should be treated as above with Digitalis followed by quinidine if necessary to establish normal rhythm and then maintenance doses. Quinidine (about one gram per day in divid doses) should be continued.

(b) If flutter recurs in the presence of orga heart disease, Digitalis should be used and sho be continued to prevent further attacks, and t is so even if fibrillation supervenes and remains. controlled fibrillation is probably nearly as efficie a cardiac rhythm as is normal rhythm.

IV. Auricular Fibrillation

A complete irregular rapid auricular activ without definite auricular contraction, with i pulses of varying strength, source and frequen reaching the A. V. Node and resulting in co pletely irregular ventricular contractions.

Mechanism: Is said to be irregular circus mov ment in the auricles.

Etiology: In order of frequency (White):

(1) Mitral stenosis and thyrotoxic heart disea

(2) Hypertension and coronary heart dis ase.

(3) Aortic valvular disease, congenital defec syphilitic aortitis, bacterial endocarditis.

(4) About 8% show no demonstrable hea disease.

(5) Other factors—sex incidence is 70% ma and there is an increasing incidence with age.

Symptoms: Generally only those of the unde lying disease, otherwise palpitation and consciou ness of irregular action of the heart.

Signs: "Characteristis sign is absolute irreg larity of the beating of the heart." (White).

Diagnosis: By above sign and the exclusion other arrhythmias as above—especially extr systoles and flutter. A pulse deficit is not a dia nostic sign but a large deficit (20+) renders fibri lation most likely. The E.K.G. is diagnostic.

Prognosis: (1) With heart disease the prognos is that of the underlying heart disease.

(2) Transient paroxysms without underlyi heart disease cause little or no disability and eve if permanent and controlled by Digitalis seems n to interfere significantly with life or activity. It the tachycardia not the arrhythmia per se that the serious feature.

Treatment: (1) Digitalis is indicated if there serious heart disease or congestive failure. It do not stop the fibrillation but acts beneficially b controlling the tachycardia which results from t fibrillation.

(2) In cases of fibrillation without heart disea or those in which there is not gross congestive fa ure, cardiac enlargements, disorders of conducti or history of embolic phenomena quinidine therap should be considered; 2/3 of such cases will be re stored, at least temporarily (few months) to norm rhythm—and if this is to occur it will do so in 2 days. The dose is as previously mentioned.

normal rhythm does not ensue, digitalis should be used for one week or more and then quinidine tried again. It is to be noted that as quinidine slows the circus movement the A.V. conduction improves and the ventricular rate may increase. This is of no concern unless it exceeds 130-140 per minute in which case Digitalis should be given and Quinidine stopped.

(3) Prevention of the arrhythmia is undertaken by the same measures as mentioned for prevention of paroxysmal tachycardia, viz: removal of possible exciting factors such as excitement, fatigue, sudden effort, heavy meals and excesses of all kinds plus correction of any associated local or general diseases plus use of quinidine if necessary.

Additional Notes on Digitalis and Quinidine

Toxic effects of Digitalis include various degrees of heart block down to and including complete heart block and an idio-ventricular rhythm; multiple extra-systoles which may go on to bigeminal rhythm and even ventricular tachycardia and ventricular fibrillation. Also, of course, the usual vomiting, diarrhoea and yellow vision.

Indications for Therapeutic Digitalis

1. Congestive heart failure from whatever cause with or without abnormal rhythm; in the presence of degrees of congestive failure needing Digitalis there is no contra-indication.
2. Rapid ventricular rate from auricular flutter or fibrillation, and rarely in that of the paroxysmal tachycardia—where quinidine is not being used (see use of quinidine under these arrhythmias).
3. Possibly as a prophylactic in patients with serious heart disease and cardiac enlargement who are not yet in failure but in whom failure may be early anticipated.

Contra-indications usually mentioned include acute myocardial infarction, various degrees of heart block, Adams-Stokes Syndrome, etc., but these are all relative contra-indications and in the presence of congestive failure lose their significance. Digitalis intoxication and multiple ventricular extra-systoles are of course contra-indications.

Quinidine Sulphate

Idiosyncracy, as shown by tinnitus, deafness, urticaria, nausea, vomiting and diarrhoea after an oral test dose of 3 gr. is rare. More serious toxic effects include ventricular tachycardia as previously mentioned and intra ventricular block (E.K.G. diagnosis); also persistent auricular flutter may appear and very rarely Sudden Death presumably from cardiac standstill (paralysis of S.A. and A.V. Nodes).

Embolism is always a concern when quinidine is used, but recent studies discount this. Askey states that 15-20% of non-quinidine treated fibrillation cases show embolism—while only 4% of quinidine treated cases do. The catch is the quinidine cases seem to occur at the time of treatment and are rather more impressive. Askey considers that failure, long standing fibrillation, enlarged heart and conduction defects are only relative contra-indications to Quinidine. The indications can be gathered from its use in the arrhythmias mentioned above.

Curtasal vs. Neocurtasal

In the January, 1948, issue of the Review under Cardiology, mention was made of the product "Curtasal," by Dr. Ben H. Lyons in his article, "Hints on Diagnosis and Management of Cardiac Disorders." In a recent letter received from Winthrop-Stearns Inc., they advised us as follows: "We discontinued the sale of Curtasal a couple of years ago, for the very reason that it contained sodium. Our new product **Neocurtasal is Sodium Free** and is intended for use in Sodium Free Diets such as those suggested by Dr. Lyons.

We requested Dr. Lyons to comment on the above. He stated "The reason for emphasizing the nature of Curtasal was that at the time the article was prepared, Curtasal was available at some pharmacies and hospitals. The new product, **Neocurtasal**, was a satisfactory salt substitute which he frequently prescribes. He regretted not mentioning this in his article.

Recent Registrants With the College of Physicians and Surgeons of Manitoba

*Brown, Ian Hay, Selkirk, Manitoba.
Cates, Bruce, A., Edinburgh, Scotland.
*Collings, Joseph Silver, Swan River, Manitoba.
Diehl, Garth Ross, 8 Edwin Apts., Winnipeg, Man.
Moore, Allin Herbert, Middlesex, England.
*Howes, E. W. M., Deer Lodge Hospital, Winnipeg, Manitoba.
*Hubar, Maurice, 1025 Henderson Highway, Winnipeg, Manitoba.
Kachuk, Henry Andrew, Sioux Lookout, Ontario.
*Kennedy, David McMaster, Fisher River Indian Hospital, Manitoba.
*McDougall, Ruth, Ste. Anne, Manitoba.
Oatway, Richard Douglas, Deer Lodge Hospital, Winnipeg, Manitoba.
*Robb, Jocelyn Irene, Winnipeg General Hospital, Winnipeg, Manitoba.
Sigal, Bernhard, Gainsborough, Saskatchewan.
*Temporary.

ANAESTHESIOLOGY

Edited by D. G. Revell, M.D., Anaesthetist, Children's Hospital, Winnipeg

The Cardiovascular System and Anaesthesia

The following several papers were presented as part of a group discussion held at the monthly meeting of the Winnipeg Anaesthetist's Society, January 6. Dr. D. C. Aikenhead had members of his staff, namely, D. M. H. Huggins, L. D. Barnhouse and F. A. Walton discuss the various aspects of the problem. Dr. J. M. Kilgour, Internist, Winnipeg Clinic, was a guest of the society to take part in this discussion.

Care of the Heart Case Under Anaesthesia

Dr. F. A. Walton

(a) Sedation: This should be adequate and even swing to over-dosage, except in congestive failure.

(b) Position on Table: The best position is supine, and if the patient is fat the head of the table should be elevated above 5° to remove the weight of the abdominal contents from the diaphragm. The prone position is poorly tolerated and should be avoided where possible. Kidney and gall bladder rests should not be used as they encroach upon the lower part of the thoracic cage.

(c) Oxygenation: A high concentration of oxygen should be given at all times, and it is advisable to start its administration before commencing the anaesthetic.

(d) Local or regional anaesthesia if used should be preceded by and supplemented by a **sleeping** dose of pentothal given throughout the procedure.

(e) Induction: It is important, particularly in anginal and coronary cases, that this be quiet and smooth. Pentothal is very useful here unless definitely contraindicated due to obesity.

(f) Intubation: This should be avoided where possible. If it is done, curare should be used beforehand and the throat sprayed with 2% pontocaine after induction of the anaesthetic. The curare should be given at least two minutes before spraying the throat.

(g) Respiratory excursion must be adequate at all times, using manually augmented respirations whenever necessary.

(h) Fluid Replacement: It is very important that blood loss be replaced. Even moderate blood loss is badly tolerated and may produce a dangerous drop in blood pressure.

(i) Continuous oxygen should be administered at the conclusion of the operation until the patient is fully conscious. If cyclopropane has been the main anaesthetic and the patient is expected to rouse soon, it is often advisable to give a sma dose of sedative such as demerol 50 mgms. abo ten minutes before discontinuing the anaesthetic

Value of the Electrocardiogram Pre-operatively

A careful clinical history and examination much more useful in estimating the cardiac stat than an E.C.G. pre-operatively. However, an o casional patient with no history or signs of cardi disease will show abnormalities in the E.C.G., su as alterations in the T waves of leads 1 or 11 whi indicates serious cardiac damage. These peop are liable to sudden and unexpected death whi is obviously of interest to the anaesthetist. Th applies to older people especially, and I am ver much in favour of routine E.C.G's before maj surgical interventions. Three of my own cas have emphasized the desirability of this. One ma died from the effects of coronary occlusion tw weeks after cholecystectomy. It was considered i retrospect that his symptoms had been due to h heart all along. Another man died nine da following partial gastrectomy for peptic ulce Post-mortem showed gross evidence of old an recent coronary occlusion. Another patient pr posed for a major operation was found to b fibrillating; the ventricular rate was about 30 an only slightly irregular. This had been missed b several doctors and nurses.

The above remarks are not meant to den surgery or anaesthesia to people with serious cardiac disease but to enable one to assess bette the desirability of surgery and the prognosis i these cases. The E.C.G. is a simple, inexpensive graphic record of considerable interest and im portance, and I don't think it should be omitte in the investigation before major surgery.

Death Under Anaesthesia Due to Heart Disease

Death under anaesthesia due to heart disease is a very rare occurrence. Lt.-Col. Bishop, of th U.S. Army, reports 20,021 cases of his own wit only 6 operating room deaths. None of these could be attributed to heart disease nor to physiological disturbances. My own experience supports thi contention, as I have had only one death in th operating room in my time out of about 7,00 anaesthetics; that death was due to massive, un controllable haemorrhage. When we consider tha in a large general hospital about 42% of th patients having surgery are over fifty years of age and that 12% of the total have **demonstrable** cardio-vascular disease, it is evident that th patient with cardiac disease is at least safe while under the anaesthetic. This contention is sup

ported experimentally also; Manning, McEachern and Hall, of Toronto, showed that the death rate following ligation of a coronary artery in the anaesthetized dog was very low. If the ligature was placed under anaesthesia but not tied until the dog was fully recovered from the anaesthetic the mortality was very high. Despite the above observations it must be recognized that patients with congestive failure, recent coronary occlusion, severe angina, complete heart block, or syphilitic heart disease with aortic insufficiency, are subject to sudden death even without anaesthesia or surgery. Also there is the post-operative course to consider. My own post-operative mortality (civilian practice) due to coronary occlusion comprises two definite and two doubtful cases, which gives a mortality of .12 to .25%. The ages of the two definite cases were 50 and 60; the ages of the two doubtful cases were 76 and 79.

Vagal Reflexes Under Anaesthesia

The ubiquitous vagus and its reflexes constitute a large subject. Vagal reflexes are particularly dangerous in patients with pre-existing heart disease, as they tolerate hypoxia less well. These reflexes may be set up by numerous stimuli which include emotion, drugs, trauma, change in blood pressure, etc.

Emotion can give rise to the well-known vago-vagal reflex. The attack is ushered in by giddiness and dimness of vision, and consciousness may be lost. The heart rate may fall to 50 or 40 per minute. At the height of the attack pallor and sweating may be prominent and the systolic blood pressure may sink to 50 mms. of mercury or less.

Blood pressure controls the resting vagal tone via reflexes from the sinus and aortic nerves. A rise in blood pressure slows the heart and vice versa. A fall in blood pressure and slowing of pulse is often seen in pressure on the carotid bulb. Even slight pressure associated with turning of the head may produce an attack, especially in old people with arteriosclerosis. Operations in this region are more dangerous than they would otherwise be. The danger is greater with light anaesthesia and when a parasympathomimetic drug such as pentothal is being used. It is recommended that the carotid bulb area be infiltrated with novocaine when surgery in this area is necessary.

Trauma: Severe trauma, such as a premature incision in the very lightly anaesthetized patient, can produce serious vagal reflexes including laryngospasm, bronchospasm, and cardiac slowing, or even asystole. Likewise intubation in the lightly anaesthetized patient often produces severe respiratory and cardiac reflexes. The respiratory reflex consists of expiratory spasm with cyanosis —this is due to severe depression of the respiratory centre by vagal afferent stimuli combined with a tremendously exaggerated cough reflex. Heart reflexes have been shown to be very common following intubation under cyclopropane and ether anaesthesia, as high as in 33% of cases. These include bradycardia, prolonged P-R interval and escape extra systoles of auricular and ventricular origin. This is often associated with markedly lowered blood oxygenation.

Drugs: Morphine sensitizes the vagal reflexes and should probably be avoided if they are expected or if these results might be more than ordinarily dangerous, as in patients with heart disease.

Atropine effectively diminishes or abolishes vagal reflexes. It is thus an essential and important premedicant for all general anaesthetics.

Scopolamine or hyoscine has similar actions to atropine but they are not as pronounced on the heart and bronchial musculature. It is probably better to use atropine if vagal reflexes are expected to be prominent.

Curare effectively depresses or abolishes vagal reflexes by action in the synapse between pre- and post-ganglionic vagal fibres. It should thus be used in cases where one might reasonably expect vagal reflexes, such as before intubation, in anaesthesia for people with active cough reflexes, and for intrathoracic operations. I have never seen severe laryngospasm when a patient has had even a moderate dose of curare. Curare may be used in the treatment of laryngospasm also. This may be largely due to its effect on striated muscle of course, but I have not seen, or at any rate recognized, any cardiac vagal effects in a curarized patient either. Curare is especially useful in conjunction with pentothal, as laryngospasm can be very severe and even fatal with pentothal anaesthesia.

Another method of abolishing vagal reflexes is by procaine infiltration of the vagal trunk in the chest (intrathoracic operations) or in the neck. In the neck one injects 60-80 c.c's of ¼% procaine deep to the point where the sternomastoid is crossed by the external jugular vein.

Cocainization or pontocainization of the throat and larynx and trachea is especially useful in abolishing vagal reflexes from this area.

Hypertension and Hyperthyroidism

Dr. D. Barnhouse

The pathology resulting from hypertension is primarily a development of spastic thickening of walls of arterioles throughout the body which gradually becomes organic. Secondarily myocardial weakness and failure develop as a result of increased myocardial strain with premature development of degenerative changes in large arteries which occasionally result in rupture or thrombosis. Serious lesions of the kidney may develop. Death is most commonly from myocardial failure but

occurs frequently from cerebral vascular accident and less frequently from renal insufficiency.

If hypertension is uncomplicated these patients are not increased risks for anaesthesia, provided the anaesthetic is skilfully given, as anaesthetic agents decrease the blood pressure and therefore decrease the strain on the heart and shock is less likely to develop. If the case is complicated by decompensation or heart lesions the anaesthetic risk then becomes the risk which is involved in dealing with such a disease.

The seriousness with which hypertension must be considered will depend on the physical state of the patient, the operation contemplated, the type of anaesthesia selected and the availability of restorative measures such as oxygen and intravenous fluids, particularly blood.

Trouble usually results from complications of the vascular tree such as shock or overaction of a drug used for or with the anaesthesia. The principal cause of increased blood pressure during anaesthesia are asphyxia, increased adrenalin in blood (poor induction) and accumulation of C02. The results of increased pressure are excessive bleeding, cerebral accidents, coronary thrombosis and congestive failure. The latter three of these accidents do not usually occur during anaesthesia but a few days or weeks later.

The choice of anaesthetic must be made with due regard to site of operation, temperament of surgeon, patient, and anaesthetist and the availability of anaesthetic drugs and restorative measures. We feel that local or regional block (without adrenalin) plus dilute pentothal drip is first choice in many cases followed in close order by pentothal, nitrousoxide, cyclopropane, ether and ethylene and finally spinal.

In the management of a case the following are essentials: thorough acquaintance with patient to induce serene confidence, rapid smooth induction, smooth level anaesthesia and rapid smooth recovery. During the operation retraction and traction on parts must be avoided, large bleeding areas must not be exposed, the patient's position must not be changed (e.g. trendelenberg), the operation must be short, an hour or less, the anaesthesia must be light and there must be plenty of blood and oxygen used during and following the operation. Analeptic drugs must always be available, particularly in those operations, e.g. sympathectomy, whoch are done as a cure for the hypertension.

Hyperthyroidism does not now present the problems to anaesthetists that it did formerly. With the introduction of thiouracil and propylthiouracil the patients now come to the operating theatre in excellent condition.

Auricular fibrillation which cannot be cont led by digitalis or quinidine is not considere contra-indication to general anaesthesia if adm istered by a competent anaesthetist.

Intravenous Procaine

Intravenous procaine is now used by an thetists in dilute solutions 0.1% or 0.2% for pa ful dressings such as burns. One-half to one gr with a similar quantity of pentothal to offset excitant effect of the procaine, is used. In addi intravenous procaine is used during anaesthesi similar strengths or in 5 or 10 cc doses of 1% 2% Novocaine to diminish the hyperirritabilit cardiac conducting system which occurs un anaesthesia with cyclopropane or accompanied asphyxia. The procaine acts by reverting a shif pacemaker to the sinus node and so establishe normal rhythm.

Anaesthesiological Considerations o Valvular Heart Disease

Donalda M. H. Huggins, M.D.

During the course of a year's work in the An thesiology Department of any of our hospitals, come in contact with a large number of patie at the various extremes of age, and with vary complications accompanying the complaint which the contemplated surgical procedure is be undertaken. Quite frequently when one visit patient before operation his first query will "Do you think my heart can stand an anaesthe or in the study of the patients chart a cardi lesion will be noted. So it behooves us all have clinical understanding of these cardiac c ditions, to better aid us in the choice of anaesthe agents, considering the pharmacological reacti of the various drugs, and their use in abnorm cardiac states, and the management of the ana thetic course, including pre- and post-operati care.

Acute Rheumatic Fever: Might conceivably complicated by the needs for an emergency ope tion. Certainly no other surgical interven should be attempted. If possible regional ana thetic would be the choice. Sauer[1] states th acute rheumatic patients do not stand surgery w and when an absolute emergency arises local spinal when applicable are the safest.

Valvular Lesions: Lewis[2] states that "T dangers involved in anaesthetising and in oper ing upon patients suffering from cardiac disea have been exaggerated in the past." When consider valvular defects in patients for surge we must assess the patient as a whole, let th tell you his story, his ability to do the ordina things of life and his response to these acts, amou of dyspnea he has, etc., on climbing stairs, walki fast, running for the bus, working daily at his j

and his amusements. These tell the story of his cardiac reserve as well as the many tests that might be carried out in an office or hospital.

Electrocardiographic studies are also of value, and I need only mention general factors such as age, hemoglobin, sedimentation rate, blood pressure, and urinalysis each with its own significance but important in fitting in the jig saw puzzle type of picture that some patients present.

Habits of an individual also play a role in his risk for surgery. Use of tobacco and alcohol in excess are detrimental to the cardiac, probably in a greater degree than to an otherwise normal individual. Toxic states, diabetes, and obesity are also prominent features in summing up the risk, as well as liver function and renal sufficiency. Chest X-ray shows size of heart and also any lung pathology. According to Levine[3] vital capacity is one of the most valuable measurements in estimating cardiac efficiency, and he says that they are much more valuable in estimating prognosis than E.K.G studies. It is one of the first changes in congestive failure, where V.C. is reduced due to engorgement of the pulmonary vessels. Levine[3], in discussing the patient with Heart Disease as a surgical and Obstetrical risk says that anyone who has been able to walk moderately without discomfort is subject to no greater hardship in undergoing an operation. So that he sums up that patients with organic Heart Disease, who are well compensated in general stand operation and anaesthetising well.

Dry[4] in an article on reducing the risk of operations for patients with cardiac disease remarks that any surgical procedure, no matter how trivial, carries with it a certain degree of risk, and that the risk is definitely greater in patients with organic heart disease, compensated or otherwise, and that mortality and morbidity rates are good because of recognition of the dangers inherent and prevention of possible sources of dangers. Adams and Lundy[8] in dealing with the subject Anaesthesia in poor surgical risks felt that provided compensation is adequate and cardiac reserve satisfactory, cardio-vascular patients stand operation and anaesthesia remarkably well. But risks increase in proportion to curtailment of activities because of symptoms. Woodridge[5] made the statement that valvular Heart Disease and Auricular Fibrillation uncomplicated by failure, do not add appreciably to the anaesthetic or surgical risk.

Congestive Failure: If congestive failure is present, Levine[3] gives a morbidity rate of 14% so cardiac decompensation adds considerably to the risk. Hermann and Hermann[6] think that C.H.F. must be considered as definitely contra-indicating surgical treatment in anything but the most urgent emergencies. If immediate operaton is necessary, pre-operative preparation must be looked after carefully. Rapid digitalization started and phlebotomy may be done, saving blood to give to patient later[7]. Adequate oxygenation is very necessary for the tissues and must be carefully looked after. Insufficiency, advanced and uncompensated, is one of the greatest complications according to Adams and Lundy[8].

Choice of Agents: Lewis[2] feels that ether is well tolerated by the young and middle aged, but is better avoided in the older age group, who are apt to be bronchitic. Quick induction is also helpful, thus avoiding struggling. Adams and Lundy[8] take each anaesthetic agent and set down its advantages and disadvantages. In summary, commenting that in surgical risks combinations of agents are often better. It is hard to lay down stiff and fast general rules of choice of agent, but choice should be one that will produce the least deleterious effects in the light of existing pathological processes, and which at the same time would be adequate for the anticipated surgical undertaking. So with the proper agent, skilfully administered, with adequate pre- and post-operative care the patient will have an improved status and his risk will be lessened.

Dry[4] reviews measures for reducing surgical risks in cardiacs and lists three factors:

(1) The internist and his preparation of the patient.

(2) The anaesthetist, providing skilled anaesthesia, recognizing the danger signs early and using sense to avert a tragedy.

(3) The surgeon, who recognizes the patients condition, limits surgery, easy manipulations and avoids blood loss.

Close co-operation between all attendents is needed. So choice of agents is practically unlimited for the Valvular Defects. Regional, where indicated, Subarachnoid Block when of value, Cyclopropane alone or in combination with other agents is good in spite of its Cardiac phenomena because these can be prevented or controlled when ether vapor is added to the mixture, as Burnstein showed, or the preventative or therapeutic use of I.V. Procaine. Nitrous Oxide, Pentothal and Curare mixtures are also good as long as anoxia is prevented.

When Decompensation is present, emergency anaesthesia requirements should be covered by some form of Regional Anaesthesia if at all possible, otherwise each case on its own merits, and the wise judgment of the anaesthetist.

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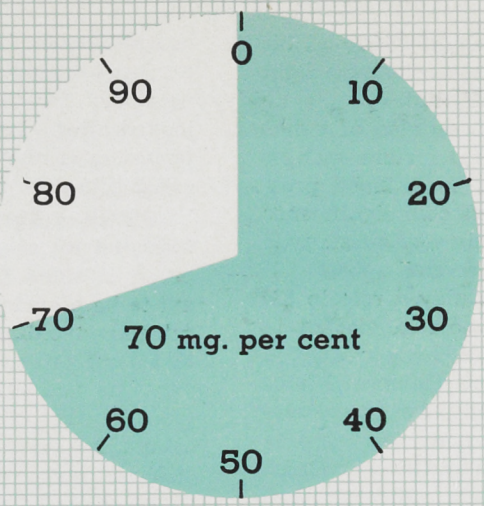

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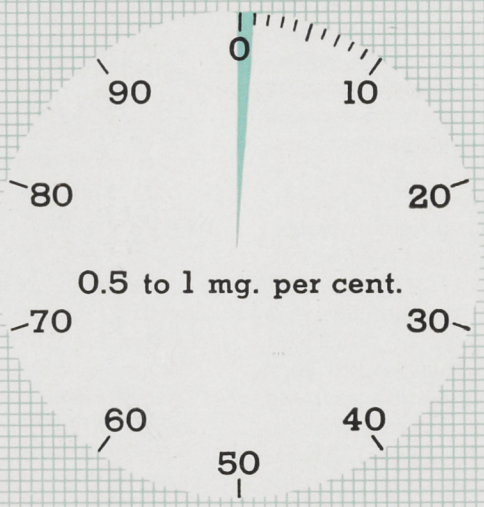

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Hospital Clinical Reports

Reported by J. M. Whiteford, M.D.

St. Boniface Hospital

Reported by F. G. Stuart, M.D.

Diagnostic Conference, January 19, 1948

Case No. 1

Mr. R. Mc. S.B.H. 47-15, 699. Male, age 48. Admitted 1.30 p.m., November 24, 1947.

This patient was struck by a switch engine and immediately brought to hospital. He complained of pain across the lower thorax on respiration.

Physical examination revealed a conscious patient in no obvious distress. B.P. was 115/60, pulse 70, and respiration 22. Tenderness was elicited on palpation over the outer end of the left clavicle, across the lower thorax and upper abdomen, especially on the left side and over the dorsal vertebrae. There were no abrasions. The abdomen was tympanitic.

In order to determine the extent of bony injury, X-ray examination of the thorax was carried out. The eighth to twelfth ribs on both sides were fractured without displacement. The right first and left second lumbar transverse processes and the outer end of the left clavicle were fractured, but not displaced. Gaseous distension of the stomach and small intestine was noted, indicating an inhibitive ileus.

Because of the site of trauma rupture of the left kidney and spleen were considered possible. A catheterized specimen of urine was obtained and was found to contain blood. An intravenous pyelogram was done, but the urinary tracts failed to visualize. This was attributed to the development of shock during investigation.

Supportive therapy was instituted and consisted of morphine for pain, gastric suction for the ileus and blood transfusion for shock. During the first night in hospital the patient continued in shock with the blood pressure being about 85/55. However, he quickly recovered and by morning the B.P. was 120/84.

He was unable to voluntarily void until the second night. Catheterized specimens obtained during the interval were sanguinous. This continued after bladder control returned.

For about four days ileus was present and rather distressing. Prostigmine and gastric suction provided some relief.

On the second day a troublesome cough developed and rhonchi were heard in both bases. Penicillin was administered.

On December 5th a retrograde pyelogram was done on the left side. This revealed considerable extravasation of the contrast media into the lower pole of the left kidney and was considered evidence of rupture of the kidney.

By the 7th of December the urine had become clear. Convalescence proceeded satisfactorily with continuation of treatment for secondary anaemia and respiratory symptoms.

An intravenous pyelogram was done on December 20th, 1947. The tear in the left kidney now appeared smaller. On the 29th of December visualization of both urinary tracts appeared quite normal. He was then discharged from hospital.

Discussion

At the time of admission the finding of blood in the urine indicated kidney rupture. The absence of evidence of peritoneal irritation i.e. rigid abdomen tended to exclude splenic rupture. Although the patient went into shock after admission there was no evidence of excessive renal bleeding. Accordingly a conservative attitude was adopted. It was stated that if peritoneal irritation or a rapidly developing fullness in the left flank had appeared, serious bleeding would have been suspected and nephrectomy would be considered. The patient's condition did not deteriorate during the first 12 hours after injury and rapidly improved thereafter. This indicated that he was not losing more blood than he could afford.

Criticism was levelled at the advisability of doing a retrograde pyelogram as soon as two weeks after the injury. It was felt that the introduction of a catheter and opaque media into a ruptured kidney was courting the risk of infection. As long as the patient was recovering radiographic demonstration of the laceration was immaterial.

In rebuttal it was pointed out that, at least in this case, no untoward complications followed the retrograde examination.

A point of interest noted radiographically and clinically was the persistent inhibitive ileus. It was generally agreed that this was commonly associated with injuries such as this patient's.

Case No. 2

Master P. L. S.B.H. 414. Male, age 13. Admitted 5.30 p.m., Dec. 16, 1947.

This lad fell beneath a truck while trying to "catch a ride." While in a supine position the rear wheel of the truck passed across his pelvis. He was immediately brought to hospital, complaining of being unable to void. Very little pain was complained of.

Physical examination revealed bruising over the left ilium posteriorly. Tenderness was elicited on lateral compression of the pelvis. Local tender-

ness was found over the right pubic bone. Shock did not seem to be present.

Immediate X-ray examination of the pelvis revealed a subluxation of the pubic symphysis with the right pubic bone ½" lower than the left. A small chip fracture from the left ala of the sacrum at the sacro-iliac joint was present.

Because of the distortion at the pubic symphysis injury to the bladder or urethra was immediately suspected. A urethral catheter was passed because he could not void. Some impediment to its passage was encountered just before the tip entered the bladder. This suggested rupture of the posterior urethra or bladder neck. About 10 cc. of urine was obtained. A Foley catheter was subsequently passed and left to drain the bladder.

The next morning further investigations were carried out. An air cystogram was done. Interstitial emphysema was noted in the retroperitoneal tissues to the right of the bladder in the space of Retzius. A moderate swelling of the soft tissues in the same locality was displacing the bladder to the left. The latter appeared to be intact. A left lateral decubitus film showed no escape of air into the peritoneal cavity. An opaque cystogram was obtained shortly after in the course of an intravenous pyelogram. The bladder appeared normal. None of the opaque media was seen in the space of Retzius, which still contained air on the right side.

The evidence at hand was now reviewed. Air and opaque cystograms revealed an intact bladder, but nevertheless air which had been introduced into the bladder found its way into the space of Retzius. How did it escape?

Three things suggested a rupture in the posterior urethra:

(1) The displacement of the pubic bones indicated that there must have been a severe shearing strain on the urogenital diaphragm. It was reasoned that this probably caused a rupture of the urethra in the membranous portion or at the apex of the prostate.

(2) The impediment to the catheter in this portion found shortly after injury.

(3) How else could the air escape since the bladder was intact? It was suggested that the air injected into the bladder, being under some pressure tracked back along the catheter to the site of rupture and then into the retro-peritoneal space. The soft tissue swelling was believed to have been caused by extravasation of urine before catheterization and probably some bleeding. The bulbous and anterior urethra were intact as no urinary extravasation was present in the anterior abdominal wall. Therefore by exclusion the rupture was localized to the posterior urethra.

The question of its extent was next considered. The intravenous cystogram showed no leakage of the opaque media which was considered evidence that there was no further extravasation into the space of Retzius and that the Foley catheter was draining the bladder adequately. Surgical drainage of the space of Retzius was therefore not indicated as no more urine was passing into it.

Irritation by the indwelling catheter caused some cystitis, but this was controlled by penicillin within a week. He was discharged 21 days after injury in a plaster cast without urinary symptoms.

Discussion

The potential danger of air embolus with an air cystogram was raised. An alternative method of detecting bladder rupture was suggested. It consists of injecting 2 or 3 ounces of sterile water through a catheter. If this cannot all be immediately recovered on withdrawal of the piston in the syringe there is a rupture of the bladder with leakage.

Almost invariably rupture of the bladder or urethra calls for supra-pubic drainage. Urine in the tissues seems to be very irritating. In this lad the rupture of the urethra did not seem to be associated with enough leakage to require drainage. The subsequent history justified the conservative course.

These two cases serve to illustrate some points concerning rupture of the urinary tract.

Rupture occurs where the urinary tract is relatively fixed i.e. at the upper or lower ends in association with trauma about the lower ribs and symphysis pubis respectively. Kidney rupture generally does not require surgical intervention unless excessive bleeding is evident. On the other hand bladder rupture is a definite indication for suprapubic drainage. This is because of the deleterious, toxic effects of interstitial urine. Occasional exceptions to this rule occur and subsequent events proved that Case 2 was one of them.

Retrograde injection is contra-indicated in kidney rupture in the early stages because of the danger of introducing infection. In bladder rupture, however, it is a valuable aid in diagnosis. The usually associated suprapubic drainage of the latter reduces the danger of subsequent infection from this procedure.

Recorded by F. G. Stuart, M.D.

Winnipeg General Hospital

Reported by J. M, Whitehead, M.D.

Streptomycin in the Treatment of Tuberculosis

Dr. J. D. Adamson

Case History

Miss M. J. H., age 21, Pupil Nurse.

1944—Tuberculin negative.

January, 1945—Started Nurses' training. Chest plate negative. Mantoux test positive.

July, 1945—Chest plate negative.

March, 1946—Catarrhal jaundice.

May, 1946—Taking affiliated course at Winnipeg General Hospital.

September, 1946—Chest plate negative.

November 13, 1946—Received influenza vaccine.

November 20, 1946—Onset of headache and patient was easily fatigued.

November 24, 1946—Developed sore throat.

November 25, 1946—Felt so rundown that she reported sick. Admitted to General Hospital. X-ray studies of the chest showed a peripheral pneumonitis of unknown origin. Sputum negative. Received palliative treatment.

December 9, 1946—Chest plate showed no change—Penicillin.

December 11, 1946—Sputum positive twice. (3 weeks after onset).

December 13, 1946—Admitted to Central Clinic.

December 17, 1946—Temperature elevated to 102° - 104°.

December 24, 1946—Chest plate—spread of infiltration.

January 2, 1947—Put on full bed rest.

January 24, 1947—Right phrenic crush.

January 27, 1947—April 8, 1947: Pneumoperitoneum.

January 29, 1947—Admitted to St. Boniface Sanatorium.

February 10, 1947—Temperature 98-103, pulse 80-130. Haemoglobin 65%; blood sedimentation rate index plus 60. Sputum positive for T.B. (Gaffky 3). Present weight 121 lbs. Usual 145 lbs. Cough and expectoration fairly marked in amount.

February 14, 1947—Diarrhoea.

February 19, 1947—Mass of glands left side of neck (ant. and post.). Mass in posterior pharyngeal wall.

February 24, 1947—Streptomycin commenced (grams 1 daily).

March 1, 1947—(One week after Strep.) Temperature suddenly improved and throughout the month remained at 100 or less. Pulse continued fast (up to 130) till March 19th, after which it stayed below 100.

March 8, 1947—X-ray film.

April, 1947—Temperature and pulse remained normal for first half of month and then gradually became higher.

May, 1947—Temperature up to 101 and pulse up to 130 throughout.

May 15, 1947—Streptomycin discontinued.

May 17, 1947—Pus aspirated from cervical glands.

June 6, 1947—350 ccs. clear yellow fluid aspirated from right thorax—positive for T.B.

June 7, 1947—Much tachycardia. Temperature dropping.

June 16, 1947—Died.

This case illustrates that streptomycin is not a cure for tuberculosis but that it does have a suppressive effect on the progress of tuberculous infection. Certain general considerations emerge from a study of such cases:

(1) The more acute the process, e.g. meningitis or miliary tuberculosis, the more effective is streptomycin and the more urgent is its early use.

(2) In the ordinary cases of tuberculosis the same general indication holds true, i.e., the more acute the infection the better the result to be expected from streptomycin therapy.

In a recent study of 225 cases treated in the United States with streptomycin, approximately 85% showed a recession of the acute exudative lesion, while approximately 8% became worse. Of those showing improvement, 17% have since shown secondary regression.

Why not give streptomycin in all cases of tuberculosis?

(1) The treatment is very expensive. Treatment at a dosage of 2 grams daily for 100 days is an average course of therapy, costing at present in the neighborhood of $700.00.

(2) Streptomycin is toxic and may produce permanent damage, notably in the vestibular portion of the 8th nerve.

(3) The organisms of tuberculosis ultimately develop resistance to streptomycin, so that at the end of a course of treatment they are still present but now resistant to therapy. This may be the result of a general effect upon all organisms or may be due to the elimination of specific strains which are more sensitive to the drug. The possibility of the development of a more virulent strain of tuberculosis should be borne in mind. Therefore, streptomycin probably should not be used in cases of minimum severity and should not be considered a substitute for present modes of treatment.

Streptomycin is useful in several other types of local tuberculous lesions, notably, tuberculous laryngitis where it may be used effectively both systemically and in aerosol form; sinuses of tuberculous origin will heal relatively quickly with streptomycin therapy and, thirdly, bilateral renal

tuberculosis with bladder frequency, etc., may be afforded symptomatic relief.

Question: Why was pneumothorax not used in this case?

Answer: It has been the experience that pneumothorax instituted in acute progressing infection, such as this, frequently results in development of empyema.

Fungus Infection of the Lungs Two Cases

Dr. C. B. Schoemperlen

Fungus infections of the lungs were first commonly described about fifteen years ago, and three main groups were distinguished, i.e. (1) yeast (e.g. monilia); (2) moulds (e.g. aspergilium); (3) higher fungi (actinomyces).

Organisms of this type are commonly present in the throat and associated passages and are not usually pathogenic but may become so, and are relatively frequent secondary invaders in disease processes of the lungs.

(1) Case History

Male, aged 37.

May, 1941—Enlisted in Canadian Army. Chest plate negative.

October, 1941 to February, 1946—Overseas in Italy, England, France, Belgium and Holland.

1943—Left dry pleurisy. Treated with sulfonamides. Intermittent cough and expectoration since.

October, 1946—Recurrence of cough, expectoration and malaise.

April, 1947—Travelling Clinic X-ray—Selkirk, Manitoba, showed right upper lobe pneumonia. Treated with sulfadiazine and improved.

July 19, 1947—A repeat X-ray showed some remaining infiltration in the right upper lobe.

July 24, 1947—X-ray, Central Tuberculosis Clinic, showed this infiltration to be clearing. Sedimentation rate normal; cough and expectoration persisting.

September, 1947—Cough and expectoration, weakness, loss of weight, with sweats.

December 6, 1947—Haemoptysis for one day.

Admitted to Deer Lodge Hospital. Sedimentation rate 104. White blood count 14,000—86% polymorphs., 12% lymphocytes, and 2% monocytes. Haemoglobin 84%. Five sputi negative for T.B. Guinea pig inoculation negative for T.B. Tuberculin positive 1/10,000. Histoplasmin negative.

December 23, 1947—Bronchoscopy—recovered purulent discharge, chiefly from the right upper lobe, and saline washings were positive for actinomyces.

December 27, 1947—Sedimentation rate 64. Started treatment of penicillin, 50,000 units o.h. 3, potassium iodide m. 25 t.i.d., sulfadiazine and sulfathiazole.

(2) Case History

The second case presented was that of a m of 23 years, who had 2½ years service with t Navy and was discharged in September, 1945, satisfactory physical condition. On January 1948, he was admitted to Deer Lodge Hospit with a history of cough and expectoration for fi months, with progressive weakness and loss of pounds in weight. Temperature on admission w 101; sedimentation rate 6 mms.; white blood cou 3,800, 52% polymorphs., 40% lymphocytes, monocytes and 1% eosinophils; haemoglobin 80 Tuberculin 1/10,000 negative and Histoplasm negative. Smears and cultures of the sputum we positive for monilia. Bronchoscopy produc bronchial washings which showed the presence malignant cells and biopsy of an axillary lym node showed an anaplastic carcinoma probab of bronchogenic origin.

Dr. A. E. Childe discussed the X-ray finding reporting extensive bilateral exudative lesion most marked in the lower lobes, giving an over cotton wool or feathery appearance, which said to be a common finding in monilial infectio of the lung.

Pulmonary moniliasis may appear in one several forms:

(1) Chronic bronchitis, in which the X-ray m be negative.

(2) Broncho-pneumonia.

(3) A severe widespread infection. Treatme consists of potassium iodide, increasing to a max mum dosage of grs. 100 by mouth or intravenous and chemotherapy with sulfonamides and penic lin.

Dr. T. H. Williams discussed the laborato diagnosis of the infections discussed herewith a presented a description with microscopic sectio of characteristic lesions.

Tumour of the Liver

Dr. C. W. Burns

Dr. Burns presented the case of a woman, who in March, 1944, had a radical amputation a breast for an advanced carcinoma, grade 3, wi palpable axillary nodes. In November, 1947, th woman reported again complaining of a mass in t left upper quadrant of the abdomen which h appeared suddenly over the preceding two week She complained also of dyspnoea, epigastric di tress and anorexia. On physical examination s appeared fairly well; she had a smooth, mobi non-tender mass in the left upper quadrant; t abdomen was otherwise negative. There was evidence of local metastasis from the breast c

cinoma and no enlargement of cervical nodes. X-rays of the chest, spine and pelvis were negative and the blood picture was satisfactory. Barium series showed the stomach to be displaced to the right and backward, apparently as a result of extrinsic pressure.

At laparotomy the left lobe of the liver was found to contain a mass which was adherent to the gastro-hepatic omentum. The adhesions were freed and the left lobe of the liver was resected. The patient has made an uneventful recovery and feels well.

Dr. Penner reported on studies of tissue sections: The original tumor removed in 1944 showed two distinct forms of breast carcinoma, the first an infiltrating duct carcinoma, the second a vascular papillary adenocarcinoma. The secondary tumor in the liver proved to be a metastasis of the second type only.

Mediastinal Tumors

Dr. M. B. Perrin

The recent chest X-ray survey of the residents of Manitoba, conducted by the Manitoba Sanatorium Board, has resulted in early treatment of conditions other than tuberculosis. Three cases of primary intrathoracic tumor have been discovered by this means in the survey of 225,000. These were as follows:

1. A girl, 4 years of age, showed a posterior mediastinal tumor, giving rise to no symptoms. This was removed at operation and proved to be a ganglio-neuroma.

2. A girl, 15 years of age, showed a posterior mediastinal tumor, also asymptomatic. This had progressed to the erosion of three ribs and the pedicle of one vertebra. At operation this proved to be a lipoma.

3. A man showing a posterior mediastinal tumor, which at present is asymptomatic. He has not yet undergone surgery.

Dr. Perrin reported that in two years he has seen 15 cases of mediastinal tumor.

These provide an interesting study in an effort to localize and type prior to operation. These tumors may be classified as follows:

1. Tumors of the Anterior Mediastinum: Dermoids and teratomata form the commonest tumors in this area, while pericardial cysts and gastric cysts may also be found.

2. Tumors of the Posterior Mediastinum:

(a) Those of nerve tissue origin are commonest, e.g. paraganglio-neuroma and neurofibroma.

(b) Those of oesophageal origin, e.g. tumors or cysts.

(c) Those of lymphoid origin.

(d) Those arising from other tissues.

Operation for removal of such tumors is undertaken for several reasons:

1. The possibility that the tumor is malignant or may become malignant.

2. The possibility of infection developing, as in dermoid cysts.

3. The possibility of rupture of cyst.

4. Size of tumor.

Dr. Schoemperlen emphasized the usefulness of the Ascheim-Zondeck test which if positive, in the absence of pregnancy, is indicative of the presence of teratoma.

Five-Year Survival in Carcinoma of the Breast

(Winnipeg General Hospital)

Dr. K. R. Trueman

Radical operation	85
Axillary nodes not involved	32, or 38%
Axillary nodes involved	53, of 62%
Cases traced	82 out of 85
Nodes not involved	31, involved 51
5-year survival, nodes not involved	23, or 74%
5-year survival, nodes involved	15, or 29%

Survival Rates of Five Years or More Following Breast Amputations at Other Centres

% Survival With or Without Lymph Node Involvement

		Yes %	No %
1. Mayo Clinic	3126	28	72.1
2. Massachusetts Gen. Hospital	236	33.3	75.5
3. Westfield State San.		31	73
4. George Washington U.	205	51.4	80.1

Dr. Trueman drew attention to the untiring efforts of Mrs. MacDonald, of the Tumor Service, in following the cases in this series.

Acute Orchitis or Oophoritis

In the recent summer epidemic a number of adults developed orchitis or oophoritis. I am most anxious to have the blood from these patients tested for antibodies neutralizing mumps virus. Will any doctor having had such a patient please send me a Keidel tube of blood accompanied by a brief clinical summary. It will take two weeks to get the tests done.

Bruce Chown, M.D., Children's Hospital.

Medico-Historical

J. C. Hossack, M.D.

A Classical Addict

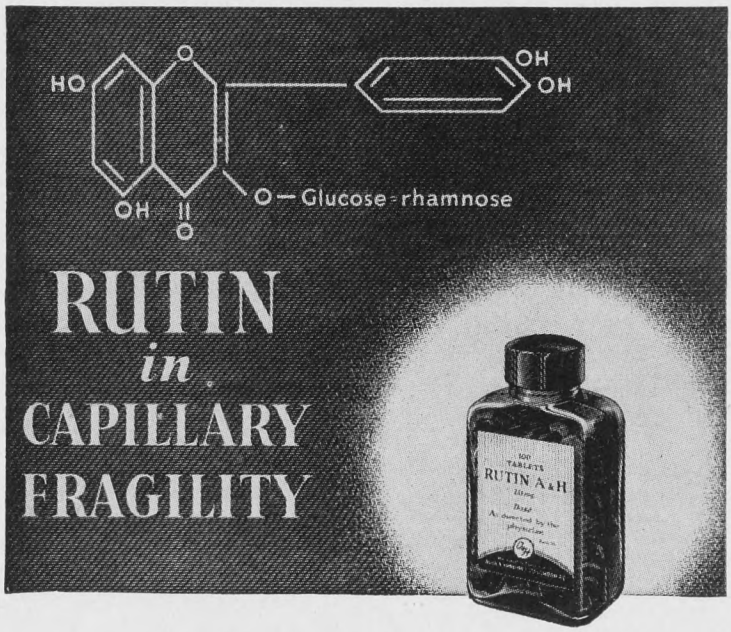
Those who have read the Confessions will have closed them with the impression that I had wholly renounced the use of Opium. This impression I meant to convey, and that for two reasons: first, because the very act of deliberately recording such a state of suffering necessarily presumes in the recorder a power of surveying his own case as a cool spectator, and a degree of spirits for adequately describing it, which it would be inconsistent to suppose in any person speaking from the station of an actual sufferer; secondly, because I, who had descended from so large a quantity as 8,000 drops to so small a one (comparatively speaking) as a quantity ranging between 300 and 160 drops, might well suppose that the victory was in effect achieved. In suffering, my readers, therefore, to think of me as a reformed Opium-eater, I left no impression but what I shared myself; and, as may be seen, even this impression was left to be collected from the general tone of the conclusion, and not from any specific words—which are in no instance at variance with the literal truth. In no long time after that paper was written. I became sensible that the effort which remained would cost me far more energy than I had anticipated; and the necessity for making it was more apparent every month. In particular, I became aware of an increasing callousness or defect of sensibility in the stomach; and this I imagined might imply a scirrhous state of that organ, either formed or forming. An eminent physician, to whose kindness I was at that time deeply indebted, informed me that such a termination of my case was not impossible, though likely to be forestalled by a different termination, in the event of my continuing the use of opium. Opium, therefore, I resolved wholly to abjure, as soon as I could find myself at liberty to bend my undivided attention and energy to this purpose. It was not, however, until the 24th of June last that any tolerable concurrence of facilities for such an attempt arrived. On that day I began my experiment, having previously settled in my own mind that I would not flinch, but would "stand up to the scratch," under any possible "punishment." I must premise that about 170 or 180 drops had been my ordinary allowance for many months. Occasionally I had run up as high as 500, and once nearly to 700. In repeated preludes to my final experiment I had gone as low as 100 drops, but had found it impossible to stand it beyond the fourth day—which, by the way, I have always found more difficult to get over than any of the preceding three. I went off under sail—130 drops a day for three days: on the fourth I plunged at once to 80. The misery which I now suffered "took the conceit" out of me at once: and for about a month I continued off and on about this mark: then I sunk to 60, and the next day to—none at all. This was the first day for nearly ten years that I had existed without opium. I persevered in my abstinence for ninety hours; that is, upwards of half a week. Then I took—ask me not how much: say, ye severest, what would ye have done? Then I abstained again: then took about 25 drops; then abstained; and so on.

Meantime the symptoms which attended my case for the first six weeks of the experiment were these: enormous irritability and excitement of the whole system; the stomach, in particular, restored to a full feeling of vitality and sensibility, but often in great pain; unceasing restlessness night and day. sleep—I scarcely knew what it was: three hours out of the 24 was the utmost I had, and that so agitated and shallow that I heard every sound that was near me: lower jaw constantly swelling: mouth ulcerated: and many other distressing symptoms that would be tedious to repeat; amongst which, however, I must mention one, because it had never failed to accompany any attempt to renounce opium—viz., violent sternutation. This now became exceedingly troublesome; sometimes lasting for two hours at once, and recurring at least twice or three times a day. I was not much surprised at this, on recollecting what I had somewhere heard or read that the membrane which lines the nostrils is a prolongation of that which lines the stomach; whence, I believe, are explained the inflammatory appearances about the nostrils of dram-drinkers. The sudden restoration of its original sensibility to the stomach expressed itself, I suppose, in this way. It is remarkable also that, during the whole period of years through which I had taken opium, I had never once caught cold (as the phase is), nor even the slightest cough soon after.

Thomas De Quincey,
"Confessions of an English Opium-Eater."

Allenburys

RUTIN



THERAPEUTIC INDICATIONS

Rutin is suggested for use in maintaining or restoring normal capillary fragility in patients with hypertension and may be of service in preventing vascular accidents; for maintaining normal capillary resistance in patients who are receiving treatment with thiocyanates, salicylates or arsenical compounds; for controlling pulmonary haemorrhage when there is no obvious cause.

Rutin Tablets A & H each containing 20 milligrammes are supplied in bottles of 100 sugar-coated tablets.

THE ALLEN AND HANBURYS COMPANY LIMITED • LINDSAY, ONT. • LONDON, ENG.

BOOK REVIEW

Nervous System

Text book of the Nervous System, a foundation for clinical neurology by H. Chandler Elliott, M.A., Ph.D. Asst. Prof. of Anatomy, Medical College of the State of South Carolina, with an introduction by Wilder Penfield, M.D., 158 illustrations, 68 subjects in color and an atlas. Montreal, J. B. Lippincott Company. $9.00.

Text book of the Nervous System, by H. Chandler Elliott has as its sub title "a foundation for clinical neurology." It is prepared for students but obviously is useful to all who wish the foundation it offers. It is a quite successful effort to simplify a difficult subject. It is written in good English. The instruction is clear. Neuro anatomy and neuro physiology are presented in association and probably with as much simplicity as is possible. The book is divided into three parts. In the first—the Nervous System in Outline—there is as as it were a low power view of the subject which orients the reader and breaks the ground. The second part fills in the outline. It goes into much greater detail but by the use of ingenious concepts and diagrams takes a good deal of the difficulty out of an admittedly difficult subject. The third part is an atlas partly photographic and partly diagrammatic. Clinical references are brought in where they are helpful but this is not a clinical text, it is a foundation for the clinical aspects. It should prove popular with all those who wish to acquire a sound basis for the clinical aspect of diseases of the nervous system.

Treatment by Diet

Treatment by Diet, by Clifford J. Barborka, B.S., M.S., M.D., D.Sc., F.A.C.P., Asst. Prof. of Medicine, Northwestern University of Medical School, Chicago; Attending Physician, Passavant Memorial Hospital; Consultant in Gastro-enterology and Gastroscopy, Diagnostic Centre, Hines Veterans Hospital; Formerly Consulting Physician, The Mayo Clinic. Fifth Edition. 14 Plates, Including 13 in Color. J. B. Lippincott Company, Montreal. $11.50.

The appalling evidence of malnutrition revealed during the war among enlistees stirred an interest in proper feeding. Disease is often favoured by this fifth column of under-nourishment and in many cases treatment is inadequate unless the diet is adjusted to the needs of the patient.

For many years the standard guide to the prescription of proper diets has been Barborka. He deals with the subject most thoroughly and the present edition (the 5th) of his book excels its predecessors in completeness.

The first part deals with diet in health and discusses the essentials of nutrition. The vitamins are considered fully in regard to their functions, the evidences of deficiency and their dietary sources. The same applies to necessary minerals. There are sections on the prevalence and the recognition of deficiency states including laboratory tests.

In the second part—The Application of Diet Therapy—are given methods of calculating and applying diets with several colored plates of comparative servings. These are accompanied by tables giving the quantities in both grams and household measures.

The main portion of the book is devoted to Diet in Disease. The diseases are classed as those in which diet is of paramount importance and those in which it is of varying importance. In each case there is a preliminary discussion of the disease state in relation to food. Thus before the tables of diet are reached one understands the why of the diet prescriptions. In the case of diabetes the introduction and diet tables cover 87 pages, obesity is given 30 pages, and peptic ulcer 24 pages. Deficiency diseases, which include the anaemias are given full consideration. The section dealing with conditions in which consideration of heart disease, hypertension, gall-bladder disease and a large number of other diseases. There is also a section in pre-operation and par-operation dietary management.

The book is of 800 pages with 14 plates, 13 of them in color. It is a complete encyclopedia on diet and diet therapy. It should be in the library or rather on the desk of every practitioner and of every dietitian. It is the sort of book that pays for itself over and over again every year. It is so completely up-to-date that no other reference is necessary for the proper and modern use of diet.

The Ego and the Mechanisms of Defence. By Anna Freud. Price $4.00. Pp. 196. New York, International University Press, 1946.

This is the first American edition of Anna Freud's psychoanalytic study of ego defence mechanisms. The original German edition was first published in 1936 by the Internationaler Psychoanalytischer Verlag under the title Das Ich und die Abwehrmechanismen. The first English translation was published in 1937 by the Garden City Press Limited, at Letchworth, Herts.

This book represents an elaboration on the theme of Sigmund Freud's account of personality

structure in terms of Id, Ego and Superego as given in his New Introductory Lectures on Psychoanalysis first published in Vienna in 1933.

The book is divided into four sections. The introductory chapter stresses the importance of ego-function in the personality and indicates that depth analysis may prove ineffective if relatively superficial functions are neglected, especially repressed portions of the ego.

The writer defines the task of analysis as follows: "To acquire the fullest possible knowledge of all the three institutions of which we believe the psychic personality to be constituted and to learn what are their relations to one another and to the outside world. That is to say: in relation to the ego, to explore its contents, its boundaries and its functions and to trace the influences in the outside world, the id and the superego by which it has been shaped and, in relation to the id to give an account of the instincts, i.e., of the id-contents, and to follow them through the tra formations which they undergo."

There is a chapter on the ego as the seat observation and one on analytic technique. rest of the first section of the book is concer with a general description of defensive operati on the part of the ego against anxiety f various sources.

The second section of the book deals in m detailed manner with the ego in relation reality, while the third and fourth sections respectively with detailed considerations of relationships with the superego and with insti tive drives derived from the Id.

This is a clearly written and well articula presentation, and although largely confined theory is precisely limited in scope. It is c cluded by a satisfactory summary and a bi index.

W. G. Glas

OBITUARIES

Reported by Ross Mitchell, M.D.

Dr. S. J. Elkin

One of the oldest graduates of Manitoba Medical College, Dr. Samuel James Elkin, of the class of 1894, died in the Winnipeg General Hospital on February 5, after a long illness.

Born at Leitrem House, Moiville, Ireland, Dr. Elkin came to Canada about sixty years ago. After graduation he practised for a few years at Emerson, Manitoba, before moving to Winnipeg. For several years he lectured on Surgical Anatomy. He was a member of the Motor Country Club, Northern Light Masonic Lodge, and the Granite Curling rink.

He is survived by his widow and one son and one daughter.

Dr. A. E. McGavin

While on his way from Carman to conduct inquest into a fatal shooting of an armed robb on the morning of Feb. 7, Dr. Andrew E. McGa died at Kane, Man., aged 72. The car in wh he was driving in company with an R.C.M.P. c stable and an undertaker was reported to h been stuck in a snowdrift a short time bef and the weather was very cold. Dr. McGa graduated from Manitoba Medical College in 19 and practised for many years at Carman wh he was held in high respect.

1948 Directory

To the members of the profession who have completed the Information Card and sent along Membership Fees, our sincere thanks.

To those who have not yet done so—your early co-operation is requested.

EDITORIAL

J. C. Hossack, M.D., C.M. (Man.), Editor

Why Not God Fathers?

For a long time I have had in mind an idea which I think is quite good and which I am determined to air. I have mentioned it to several of my colleagues and to some of the students. They all agree on its general excellence but all agree, also, that it has a fault—it won't work. That is a rather serious handicap to any plan but I am reluctant to accept this decision because I feel that the scheme has merit and might be made workable. Anyway here it is.

As you know when a boy is baptised he is given two god fathers whose duty is to supervise his spiritual development. I suggest when a boy is baptised, as it were, into the faculty of medicine he should then also be given two god-fathers. One of these would be a member of the faculty. Towards his protege he would stand in loco parentis. He would interest himself in the lad as a student but chiefly as a person. He would be ready to help and advise when academic, emotional and other difficulties arise. Frequency of contact would generate understanding on the one hand and confidence on the other. Now when a faculty member is consulted by a student both are handicapped. The one consulted has only the slightest knowledge of the youth, and the student, awed perhaps and bashful cannot unbosom himself as he would wish. An accepted relationship of protector and protege of god-father and god-son, would make for intimacy and confidence so that problems would be frankly and easily discussed and the advice would be more sound.

The academic god-father would function during the session. Naturally he would be a specialist. Between sessions the second god-father would take over and he would be a general practitioner. During the summer the student would work with and, if in the country, would live with, his god-father. He would go with the doctor on his rounds and watch the technique of examination diagnosis and treatment. The doctor would explain his procedures, discuss the conditions seen and, while instructing his junior, himself learn by teaching. At first the student would be able to help little but as the years passed he would become increasingly useful. But the student himself would profit enormously. He would find his lectures and demonstrations much more useful In the pathology lab. he would see tissues that might have come from the patients he saw in the summer. In physiology the lecturer's discussion on dyspnea, cyanosis, odema, etc., would mean much more as examples of these distresses come quickly to mind. Thus would be begotten a clearer understanding of the phenomena of disease and a greater enthusiasm in active practice. Moreover such experience in the country might overcome the present distaste to practice there.

It is likely that, when two men are interested in one boy, they will find opportunity to discuss his faults and merits. It would be logical for the three to discuss the future when the course nears its end. Then those who had such excellent opportunity to watch the lad could show him the best way for him to use his talents, the best path for him to follow. Thus one might be advised to follow research, another to aim at teaching, a third encouraged to use his skilful hands as a surgeon, a fourth to engage in general practice. So advised the student would be likely to find himself launched on the sea of life with chart and compass, conscious of his goal. At present there is too much drifting, too many taking, often from necessity the quickest way to make an income. The best work is done by men who are in their proper places and I believe that it should be the duty of an alma mater to strive in every way possible to guide the new graduate. We are so very careful about their pre-entrance history that it would only be logical to show a like interest in their postgraduate fate.

Now there is my plan. As I said before none deemed it practical and in every instance the expressed fault was the same—the members of the faculty would not be interested. Perhaps that is true of some but it is not true of all so let us see if we can't find a way to make it work. Let us eliminate academic god-fathers for those students who live with their parents and who do not feel that they need such help. Let us put it up to the students—do you want to play the role of proteges? Let us ask the Dean to indicate the students who would profit by such relationship. Let us ask the members of the faculty who among them would accept the opportunity of re-living his college days, of living up to the letter of the Hippocratic Oath, of giving what he as a student probably often wished that he could get.

There would be little difficulty with the general practitioner. I am sure that many country doctors would find the relationship profitable and they would prove enthusiastic teachers. City doctors, too, include many competent practitioners, who have the knowledge and ability to teach but who lack the opportunity. How, I am sure, they would welcome it and how useful would be their instruction.

Well, there you are, I maintain that it is a good idea. It can be modified as necessary but I be-

lieve that it would be of great advantage to the students.

Quiet Please

Hospitals are not the noisiest places in the world yet neither are they the quietest. To be sure, the din is not constant but there is enough of it to call for comment. The noises are of various sorts and sizes. There is the rare—very rare occasion when a careless maid shatters the quiet by dropping a tray-load of dishes. The effect remotely suggests the majestic crash of music which interrupts some symphonies. This, however, is definitely rare, the smashing of individual dishes being preferred to mass destruction.

The common noises are of two origins, human and mechanical. The former are heard chiefly during visiting hours when high pitched voices and shrill peals of laughter pierce the loud hum of many conversations. Few visitors, it would seem, keep their voices sweet, gentle and low. But the most objectionable form of human noises is the after-visiting-hours conversations of patients. There always seems to be a number of people who are in for "tests." These, when the vistors have gone, indulge in private visiting among themselves. They hold clinics on themselves, discover mutual acquaintances and then, about 11 o'clock they get down to the business of settling problems national and international. At mid-night most hospital inmates are much more interested in sleep than in what to do about Russia but the fact has to be stressed to the debaters before they will shut up.

The mechanical noises come from two main sources—radiators and radios. The former seem to have a remarkably large repertoire ranging from a quiet not disagreeable hum to series of staccato hammerings that, under proper circumstances give one the impression that he is in a boiler factory. The various combinations of these sounds tend to relieve the monotony of the night and certainly prevent the occurrence of hypersomnia. It is nevertheless remarkable how quickly one becomes used to the din and, indeed,, actually misses it in an extraordinarily peaceful night.

The most objectionable noises, however, emanate from radios. Wailing, shrieking, howling women always seem to be on tap. Groaning men and ancient come-backs with grating, rasping voices bleat out raucously on the turn of a dial. Then there is what passes in this degenerate age for music. Some concord of sweet sounds which has soothed generations is bedeviled by a barbarian "arranger" into a mass of discordant noise. "Annie Laurie," "Loch Lomond" and other sacred airs (to the Scots at least) are "jazzed up" by individuals who have no music in their souls. All this is forced upon unwilling ears by thoughtless individuals whose own obtunded senses being unaffected, fail to realize that not all the inmates of a ward share their perverted ideas of pleasing sounds. Had I anything to do with it I would give the possessor of each radio a card printed thus "You are given the privilege of having your radio so that the tedium of your illness may be relieved. But it should be no louder than necessary to reach your ears. If it becomes a source of annoyance or discomfort to others, the privilege will be cancelled and the machine removed." That, perhaps might make the thoughtless take heed.

Of course these gross disturbances of peace are not constant. But when they do occur they are exceedingly annoying, especially to the really sick and to those who are in the irritable stage of convalescence. When sleep is about to come there should be no hindrance to its approach. The amusement of the well should never be permitted to interfere with the welfare of the sick and it that welfare peace and quiet are above all things needful.

Note to the Social Editor

Dear Kay:

Only careless editing permitted the present in the February issue of your reference to myself. How could you so play fast and loose with my good name? Just imagine what those who do not know me will think when they see beside my name a brazen scantily clad hussy and read of me being a bongo bongoist and a boogie woogieist, I who suffer from virtue rather than from dissipation. Indeed I attribute my sufferings to my inability—Scot though I am—to follow the advice of the Apostle Paul to Timothy, "Take a little wine for thy stomach's sake and thine often infirmities." Alas I have no palate for whisky (Paul mentioned wine only because he had not heard of whisky). I believe that if I had developed a taste for what so many find a delectable fluid I might have been spared much distress. And tell me, Kay, why was it that you came not to see me, you of the pleasant smile and the mirthful conversation and the cheerful heart that doth good like a medicine? But no. You sit in your little office drawing pictures of ballet dancers who are probably no better than they should be and casting aspersions on my character. Fie on you, Kay, fie on you. How do you mean to make amends?

J. C.

CURRENT NOTES & NEWS

Reported by M. T. Macfarland, M.D.

Fee Schedule Revision

Ever since the Annual Meeting of the Association in October, when the position of the Manitoba Medical Service was reviewed at some length by the Medical Director, Treasurer, and other members of the profession who have given freely of time and effort to make the Manitoba experiment in prepaid medical care a success, there has been increased interest on the part of those who provide some of the services. The visit in November of Mr. Frank Smith, Director, Associated Medical Care Plans of the United States, further emphasized several points of difference between the Manitoba plan, and those south of the border, of which he had first-hand knowledge. Most of the latter operate on a lower fee scale, one fee only is paid for the same service whether rendered by General Practitioner or Specialist, and where the fee is below the practitioner's regular scale, and the subscriber earns more than a certain salary, extra charges are allowed. The Board of Trustees of the Manitoba Medical Service requested the formation of a joint committee representing the Manitoba Medical Association and their own body to bring in recommendations concerning the Fee Schedule which would make for more satisfactory adjustment of accounts. Two meetings have already been held, and a third is anticipated shortly. Following the complete revision the proposed changes will require ratification by the Board of Trustees of the Manitoba Medical Service, and the Executive of the Manitoba Medical Association. Any major changes will necessitate approval by the whole Association in special or regular session.

General Practitioners

The Business Manager of the Review is still searching for the "leak" which enabled an editorial dealing with the proposed organization of General Practitioners to appear as a major news item on the front page of one of the leading dailies on Friday, February 13th, 1948.

Organization of the group was formally announced in the local papers as well as over the wires of the Canadian Press, following a meeting which was held at the Medical College on Wednesday evening, February 18th.

The aims of the organization are threefold:

(1) To guard the rights of the public so that the services of the general practitioner or family doctor will not disappear.

(2) To guard the rights of the general practitioner so that the high standard of service will be maintained.

(3) To work in co-operation with all organizations of the medical profession.

Officers were elected as follows: President, Dr. Quentin D. Jacks; Vice-Presidents, first, Dr. J. Roy Martin; second, Dr. A. T. Gowron; third, Dr. A. A. Keenberg; fourth, Dr. A. G. Dandenault; Corresponding Secretary, Dr. Anna Wilson; Recording Secretary, Dr. Donald N. C. McIntyre; Treasurer, Dr. M. M. Brown. On the Executive Council are listed doctors who enjoy the privileges of the various hospitals. The group accepted the invitation of the Executive of the Manitoba Medical Association to name three members who would attend the General Council Meeting of the Canadian Medical Association, which meets in Toronto, June 21-25. The Association should benefit greatly from the added stimulus of an enthusiastic group. Perhaps some significance may be attached to the casual appearance of the oak leaves and acorns at the bottom of page 91, of the February Review, on which the matter was first discussed by the Editor.

Yellow Fever Inoculation

Under date of January 27, 1948, Dr. J. L. Lamont, District Medical Officer, Department of Veterans' Affairs, addressed to the Association office a copy of the Bulletin issued by the Department of National Health and Welfare concerning Yellow Fever Inoculation. Immunity against Yellow Fever is recommended for all persons planning to travel in areas where the disease is either endemic or epidemic. Centres where such inoculation and valid International certificates may be obtained are listed, as arranged by the Quarantine Division.

In Winnipeg, Manitoba, application should be made to Director of Pathology, Deer Lodge Hospital, Department of Veterans' Affairs. It is understood that a charge of $2.00 is made for the Official Certificate.

Winnipeg Medical Society

A special meeting of the Winnipeg Medical Society was held in Theatre "A" of the Medical College, on Monday, January 12th, 1948.

The meeting was presided over by Dr. C. E. Corrigan, President. Approximately 150 members attended. The meeting was held on the occasion of the visit to Winnipeg of Dr. Rhodes, Director of Virus Research, and Associate Director of the Connaught Laboratories, University of Toronto, and Mr. Gibbord, Director of National Research Laboratories, Ottawa. The appearance of these gentlemen was sponsored by the Hon. Paul Martin, Minister of National Health and Welfare. Mr. Gibbord spoke on the "Functions and Facilities of the National Research Laboratories," while Dr. Rhodes' subject was "Newer Knowledge of Virus Diseases."

A general meeting of the Winnipeg Medical Society was held in the Medical College on Friday, February 6th, 1948.

Dr. C. E. Corrigan requested the members to stand, in memory of the late Dr. August Blondal and Dr. S. J. Elkin. The Secretary read a letter from Dr. L. G. Bell, Chairman of the Library Committee, thanking the Society for a substantial donation to the Library Fund of the Medical College.

Dr. Wallace Wilson, immediate Past-President of the Canadian Medical Association, spoke briefly Dr. Wilson is Chairman of a special C.M.A. Committee, set up to consider the recognition of the General Practitioner. He outlined some of the discussions which had already taken place, and stated that at the Annual Meeting of the Canadian Medical Association in June, one evening would be devoted to consideration of the problems of this group. Since Dr. Wilson was to meet those specially interested in this matter the following day, no discussion of the subject took place.

The meeting was then turned over by the Chairman to Dr. A. T. Mathers, Dean of the Faculty of Medicine. Dr. Mathers expressed his regret at the inability of the President, A. W. Trueman, to attend the meeting because of illness. The Dean discussed the present aims and objectives in Medical Education. Following this, he called upon the heads of several departments to discuss the recent physical changes resulting from renovation of their departments, and to describe whatever new methods in teaching had been initiated in those departments. Those taking part in this presentation included Doctors I. M. Thompson, H. V. Rice, Daniel Nicholson, Bruce Chown and J. D. Adamson.

The further portion of the programme included a tour of the building by the members of the Society, in order that they might be shown the changes which have been recently instituted throughout the College.

C.M.A. Meeting in Toronto

In the January, 1948, issue of the Canadian Medical Association Journal (Pages 89 and 90), attention was called to the necessity of making early hotel reservations. It is anticipated that available accommodation will be taxed to capacity, so, "A word to the wise . . ."

Group Insurance

Did you know that in addition to the standing committees usually chosen each fall to carry out the routine work of the Association there was added, this year, a special committee to study and report on Group Insurance—Life, Accident and Sickness —for the doctors of Manitoba? Two meetings of the committee have already been held and suggestions from interested parties, professional or lay, will be welcomed by any member of the comm tee, which consists of Doctors L. R. Rabson, Cha man, Wm. J. Boyd, M. J. Ranosky and F. Hart Smith.

Northwestern District Medical Society

Dr. Ed. Hudson, of Hamiota, reports an inform evening meeting of the local Northwesters, wh was held early in December. Doctors W. Hames, Kenton; J. G. Pincock, Oak River; D. Stewart, Rivers; E. D. and J. E. Hudson, and nurses of Hamiota Hospital attended. At the of the same month, a farewell party was h for Dr. J. G. Pincock, who is to pursue po graduate studies at Deer Lodge Hospital. Medi discussions were barred, and the evening w purely social.

Northern District Medical Society

Due to an oversight, the last meeting of t Northern District Medical Society was not report in these columns. The matter was brought to o notice by the energetic representative of t Society to the Association Executive, who sta that in addition to the notes listed, regular me ings with case discussions are held monthly at t hospital.

A meeting of the Northern District Medi Society, held at the General Hospital, Dauph Manitoba, on the evening of Friday, Nov 28th, 1947.

Present Were: Doctors

R. E. Dicks, President	Dauphin
W. G. Ritchie, Secretary-Treas.	Dauphin
A. S. Little	Dauphin
M. Kagan	Dauphin
R. M. Creighton	Dauphin
S. W. Fox	Gilbert Plains
R. T. Watkins	McCreary
T. F. Malcolm	Swan River
R. Lyons	Winnipeg
S. Israels	Winnipeg
M. T. Macfarland	Winnipeg

1. Following the splendid goose dinner ser under the direction of Miss Mackinnon, dietiti the visitors were taken on a tour of the hosp by Miss A. Pearson, Superintendent, and M Sinclair, Instructress of Nurses.

2. An emergency Laparotomy delayed the st of the Scientific Session, which was a discussion "Treatment of Infantile Diarrhoea," by Dr. Israels and of "Common Gynaecological Op Procedures," by Dr. Ruvin Lyons.

3. At the business session a splendid accou of the last Executive Meeting of the Manito Medical Association was given by Dr. Adam Litt while a few remarks were made by Dr. M. T. Mc farland, who also moved a vote of thanks to t hosts, hostesses, and the speakers.

SOCIAL NEWS

Reported by K. Borthwick-Leslie, M.D.

Congratulations to Dr. C. H. Walton who—having attended the Annual Convention of Allergists in St. Louis in December, was elected to the Followship of the American Academy of Allergy. "Chuck" is the first Canadian to be so honored.

♦

We miss the cheerful countenance and friendly kibitzing of Dr. Hutchison at Grace Hospital. He is taking a months' P.G. work in Anaesthesiology at the Vancouver General Hospital and I believe bemoaning the rain and slush. He misses our bracing — and how bracing — Manitoba frost.

♦

A most pleasant, interesting, and instructive evening meeting of the Manitoba Medical Women was held Feb. 3rd, at the home of Dr. Jessie McGeachy. A symposium "The Highlights of Psychiatry in the Practise of Medicine," was presented by Drs. Steinhauer, Rait-McKenty, McKim, Emma Adamson and McGeachy. Lunch was excellent, too, thank you.

♦

The marriage took place Jan. 24, of S. K. Beattie to Dr. Wm. Garth Hemenway in St. Stephen's Broadway Church. After their wedding trip in Eastern Canada, Dr. and Mrs. Hemenway will reside in Rosetown, Saskatchewan.

♦

On Feb. 7, bridal vows were exchanged between Almuna Ruth Smith and Dr. Albert Leonard Nowell. The young couple left for Minneapolis and several cities in the States, before settling down in Brandon, where Dr. Nowell is a member of the staff of the Sanatorium. Dr. Nowell has recently returned from post-graduate study in Scotland. He is a Licentiate of the Royal College of Physicians and Surgeons, Edinburgh, also of the Royal Faculty of Physicians and Surgeons, Glasgow.

♦

Dr. and Mrs. Quentin Jacks announce the birth of their second son, Ronald Brian. 'Tis a big year for Quentin, he is being congratulated also, on becoming President of the newly formed "Ogpu,"—meaning the Old (?) General Practitioners Union.

♦

Dr. and Mrs. Bruce Hunter and small son, Peter, arrived by plane from Edinburgh, where Dr. Hunter has been doing post-graduate work. At present they are visiting in Reston and Minnedosa, so I haven't found out Bruce's new degrees!

Dr. A. L. Rice, a graduate of the University of Toronto, has been appointed as senior physician at the Manitoba School for Mental Deficients, Portage la Prairie. Welcome to Manitoba, Dr. and Mrs. Rice.

♦

Our old friend, Dr. Murray Clare, was elected President of the Neepawa Branch of the Game and Fish Association a short time ago. Congratulations.

♦

The engagement is announced of Lillian Ransby to Dr. Svein Octavius Eggertson. The wedding will take place March 18, in Westminster United Church.

♦

Dr. and Mrs. J. M. Huot, have left for Chicago, where Dr. Huot will take a post-graduate course in the University of Illinois.

♦

Dr. Elmer James, just returned from a vacation in the South, is looking very healthy. Apparently Vitamins, á la Orange Juice, Strawberries, etc., agree.

♦

Other lucky winter vacationists are Dr. and Mrs. C. M. Strong, who are motoring to the Southern States, and Dr. and Mrs. H. B. Sommerfeld, who are spending several weeks in Victoria, B.C.

♦

Dr. and Mrs. Alan McCarten are receiving congratulations on the birth of Rosemary Josephine, Feb. 8.

♦

Dr. and Mrs. D. M. Mitchell announce the birth of Donald Paul, Feb. 8.

♦

Dr. and Mrs. J. J. Lander announce the arrival of their daughter, Judith, Jan. 27.

♦

Dr. and Mrs. C. F. Benoit announce the birth of Shirley Elizabeth, Jan. 28. This has been a busy month for the Obstetricians!

♦

Athol and I have declared an armistice. Hats off, I just pay the checks.

♦

We extend our sincere sympathy to the family and friends of Dr. S. J. Elkin, who died recently following a prolonged illness.

♦

Feb. 7, Elma Louise Copeland became the bride of Dr. Glenn Willson, of Flin Flon, son of Dr. and Mrs. W. A. Willson. After a short honeymoon, they will return to Flin Flon to take up residence.

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1947		1946	
	Dec. 28,'47 to Jan. 24,'48	Nov. 30 to Dec. 27,'47	Dec. 29,'46 to Jan. 25,'47	Dec. 1 to Dec. 28,'46
Anterior Poliomyelitis	0	4	0	0
Chickenpox	184	229	130	143
Diphtheria	2	6	12	15
Diphtheria Carriers	0	1	3	3
Dysentery—Amoebic	0	0	0	0
Dysentery—Bacillary	0	0	0	1
Erysipelas	3	3	5	1
Encephalitis	0	0	0	0
Influenza	1	3	4	9
Measles	15	114	621	310
Measles—German	10	6	0	0
Meningococcal Meningitis	0	1	1	3
Mumps	110	86	152	139
Ophthalmia Neonatorum	0	0	0	0
Pneumonia—Lobar	7	1	13	24
Puerperal Fever	0	0	0	0
Scarlet Fever	11	15	25	38
Septic Sore Throat	0	0	1	2
Smallpox	0	0	0	0
Tetanus	0	0	0	0
Trachoma	0	0	0	0
Tuberculosis	17	98	16	146
Typhoid Fever	0	2	0	0
Typhoid Paratyphoid	0	0	0	0
Typhoid Carriers	0	1	0	0
Undulant Fever	0	0	0	1
Whooping Cough	56	85	52	53
Gonorrhoea	120	99	181	133
Syphilis	38	34	31	35
Diarrhoea and Enteritis, under 1 yr.	6	8	5	18

Four-Week Period December 28, 1947, to January 24, 1948

DISEASES (White Cases Only)	*743,000 Manitoba	*906,000 Saskatchewan	*3,825,000 Ontario	*2,962,000 Minnesota
*Approximate population.				
Anterior Poliomyelitis	....	8	....	5
Chickenpox	184	207	1814	....
Diarrhoea and Enteritis	6	....	....	....
Diphtheria	2	3	7	24
Dysentery—Amoebic	....	....	5	....
Erysipelas	3	1	4	....
Infectious Jaundice	....	....	5	....
Influenza	1	2	17	1
Encephalitis	....	2	1	1
Malaria	....	....	....	5
Measles	15	17	2052	1227
Measles, German	10	1	75	....
Meningococcal Meningitis	....	1	6	1
Pneumonia Lobar	7	....	....	....
Mumps	110	253	933	....
Scarlet Fever	11	6	333	184
Septic Sore Throat	....	1	8	....
Tuberculosis	17	44	118	156
Typhoid Fever	....	....	4	....
Para-Typhoid Fever	....	....	1	....
Undulant Fever	....	....	3	19
Whooping Cough	56	7	101	247
Gonorrhoea	120	....	243	....
Syphilis	38	....	140	....

DEATHS FROM REPORTABLE DISEASES

For 4-Week Period December 31, 1947, to January 27, 1948

Urban — Cancer, 20; Pneumonia Lobar (108, 107, 109), 1; Pneumonia (other forms), 5; Syphilis, 2; Whooping Cough, 1; Dysentery, 1; Hodgkin's Disease, 1; Diarrhoea and Enteritis (under 1 year), 1. Other deaths under 1 year, 7. Other deaths over 1 year, 93. Stillbirths, 3. Total, 103.

Rural—Cancer, 9; Influenza, 1; Pneumonia Lobar (108, 107, 109), 2; Pneumonia (other forms), 6; Tuberculosis, 3; Hodgkin's Disease, 1; Diarrhoea and Enteritis (under 1 year), 2. Other deaths under 1 year, 10. Other deaths over 1 year, 37. Stillbirths, 2. Total, 49.

Indians—Influenza, 1; Tuberculosis, 1. Other deaths under 1 year, 3. Other deaths over 1 year, 0. Stillbirths, 0. Total, 3.

♦

Chickenpox is epidemic in many parts of the province.

German Measles—If you attend a woman for confinement who has had this disease during the first three months of pregnancy we would be very pleased to hear from you as to whether the infant is normal or not. The same thing may apply generally to any virus disease.

Tuberculosis is still a definite problem in Manitoba. The surveys are doing wonderful work in discovering new and old cases. We are still somewhat short of beds and there are some cases with positive sputum still at home. When these persons cannot be admitted to sanatorium special precautions should be taken in the home to prevent spread of infection. The home should be placarded so that visitors are warned of the danger of infection.

Medical Library

Recent Accessions From October, 1946 to October, 1947

(Continued from Vol. 28, No. 2, Page 111)

New York Academy of Sciences. Antibiotics, by W. W. Duemling and others.
New York Academy of Sciences, 1946. P 31-218. (Its Annals, Vol. 48, art. 2).

New York Academy of Sciences. Blood grouping, by Wm. C. Boyd and others.
New York Academy of Sciences, 1946. 111 p. (Its Annals, Vol. 46, art. 9).

New York Academy of Sciences. Folic acid, by Y. Subba Row and others.
New York Academy of Sciences, 1946. 92 p. (Its Annals, Vol. 48, art. 5).

New York Academy of Sciences. Lymph, by P. D. McMaster and others.
New York Academy of Sciences, 1946. 203 p. (Its Annals, Vol. 46, art. 8).

New York Academy of Sciences. The physico-chemical mechanism of nerve activity, by D. Nachmansohn and others.
New York Academy of Sciences, 1946. 227 p. (Its Annals, Vol. 47, art. 4).

New York Academy of Sciences. Proteins and protein hydrolysates in nutrition, by J. B. Allison and others.
New York Academy of Sciences, 1946. 130 p. (Its Annals, Vol. 47, art. 3).

New York Academy of Sciences. Experimental hypertension, by Wm. Goldring and others.
New York Academy of Sciences, 1946. 179 p. (Its Special publications, Vol. 3, p. 1-180).

Newman, Charles. Medical emergencies; 3d ed.
Churchill, 1946. 117 p.

Nielsen, J. M. Agnosia, apraxia, aphrasia; 2d ed.
Hoeber, 1947. 292 p.

Page, I. H. Arterial hypertension, its diagnosis and treatment.
Year Book Pub. (c1945-46). 350 p.

Panton, Sir P. Clinical pathology; 5th ed.
Churchill, 1945. (1947). 450 p.

Patten, B. M. Human embryology.
Blakiston (c1946). 776 p.

Peters, J. P. Quantitative clinical chemistry; 2d ed.
Williams & Wilkins, 1946 v. 1.

Pharmaceutical Society of Great Britain. Penicillin; its properties, uses and preparation.
Pharmaceutical Pr., 1946.

Porter, J. R. Bacterial chemistry and physiology.
J. Wiley, 1946. 1073 p.

Pregl, F. Quantitative organic microanalysis, based on the methods of Fritz Pregl, completely rev. and ed. by Julius Grant; 4th ed.
Blakiston, 1946. 238 p.

Pottenger, F. M. Symptoms of visceral disease; 6th ed.
Mosby, 1944. 442 p.

Rankin, F. W. Cancer of the colon and rectum; its diagnosis and treatment.
C. C. Thomas (c1939-1945). 358 p.

Richards, R. L. The peripheral circulation in health and disease.
Livingstone, 1946. 153 p.

Richter, Victor von. The chemistry of the carbon compounds. Vol. 3: The aromatic compounds . . . newly tr. from the 12th German ed. by A. J. Mee. (3rd English ed.).
Elsevier Pub. Co., 1946. 794 p.

Rolleston, Sir H. D., and Moncrieff, A. A., eds. Modern anaesthetic practice; 2d ed.
Eyre and Spottiswoode, 1946. 150 p.

Russell, P. F. Practical malariology.
Saunders, 1946. 684 p.

Sachs, E. The care of the neurosurgical patient, before, during and after operation.
Mosby, 1945. 267 p.

Scott, W. A. The essentials of obstetrics and gynecology.
Lea and Febiger, 1946. 390 p.

Semashko, N. Public health in the U.S.S.R.
Soviet News, 1946. 48 p.

Sherrington, Sir C. S. The endeavours of Jean Fernel, with a list of the editions of his writings.
Cambridge University Pr., 1946. 223 p.

Simmons, J. S., ed. Laboratory methods of the United States army; 5th ed.
Lea and Febiger, 1946. (c1944). 823 p.

Smith, Austin, ed. Medical research, a symposium.
Lippincott, c1946. 169 p.

Snedecor, G. W. Statistical methods applied to experiments in Agriculture and Biology; 4th ed.
Iowa State College Pr., 1946. 485 p.

Snyder, Laurence H. The principles of heredity; 3d ed.
Heath, 1946. 450 p.

Soffer, L. J. Diseases of the adrenals.
Lea and Febiger (c1946). 304 p.

Soskin, S. Carbohydrate metabolism; correlation of physiological, biochemical and clinical aspects.
University of Chicago Pr. (1946). 315 p.

Spies, T. D. Experiences with folic acid.
Year Book Pub., 1947. 110 p.

Stallard, H. B. Eye surgery.
Wright, 1946. 444 p.

Steindler, A. The traumatic deformities and disabilities of the upper extremity.
C. C. Thomas, 1946. 494 p.

Stodtmeister, R. Moderne eisentherapie.
Stuttgart, Wissenschaftliche verlagsgesellschaft. m.b.H., 1943. (Ann Arbor, Michigan, J. W. Edwards, 1944).

Stevenson, L. G. Sir Frederick Banting.
Ryerson, 1946. 446 p.

Sumner, J. B. Chemistry and methods of enzymes; 2d ed., rev. and enl.
Academic Pr., 1947. 415 p.

Szent-Gyorgyi, A. Chemistry of muscular contraction.
Academic Pr., 1947. 150 p.

Tasman, I. S. The eye manifestations of internal diseases; 2d ed.
Mosby, 1946. 614 p.

Te Linde, R. W. Operative gynecology.
Lippincott (c1946). 751 p.

Thannhauser, S. J. Lipidoses; diseases of the cellular lipid metabolism, ed. by F. A. Christian.
Oxford University Pr. (c1940). 370 p.

Turner, G. G. Injuries and diseases of the oesophagus.
Cassell, 1946. 100 p.

U.S. National Cancer Institute. A symposium on mammary tumors in mice.
American Assoc. for the advancement of science, 1945. 223 p.

Vaughan, W. T. Primer of allergy. 2d ed.
Mosby, 1943. 176 p.

Weinbren, M. A manual of tomography.
Lewis, 1946. 270 p.

Weisman, A. I. The South African frog (Xenopus laevis) in pregnancy diagnosis; a research bulletin.
New York biologic research foundation, 1944. 134 p.

Whitby, Sir L. E. Disorders of the blood; diagnosis, pathology, treatment and technique; 5th ed.
Churchill, 1946. 665 p.

Whitwell, J. R. Analecta psychiatrica.
Lewis, 1946. 160 p.

Widdowson, T. W. Special on dental anatomy and physiology and dental histology; 7th ed.
Staples, 1946. 2 v.

Wintrobe, M. M. Clinical hematology; 2d ed., thoroughly rev.
Lea and Febiger, 1946. 862 p.

Wolberg, L. R. Hypoanalysis.
Grune and Stratton, 1945. 342 p.

Wright, Samson. . . . Applied physiology; 8th ed.
Humphrey Milford, 1945. 944 p.

New Journals and Serials Added to the Library

American Medical Women's Association Journal.
V. 1 (1946).

American Practitioner (monthly). Successor to "Clinics."
V. 1 (1946).

Archives of Physical Medicine (monthly).
V. 28 (1947).

Gastroenterology (monthly).
V. 1 (1943).

Human Relations (quarterly).
V. 1 (1947).

Plastic and Reconstructive Surgery (bi-monthly).
V. 1 (1946).

Postgraduate Medicine (monthly). Continuation of Interstate Postgraduate Medical Assoc. of North America; Proceedings. (Annual).
V. 1 (1947).

Quarterly review of psychiatry and neurology (quarterly).
V. 1 (1946).

Recent progress in hormone research; proceedings of the Laurentian hormone conference.
V. 1, ed. by Gregory Pincus. v. 1 (1947).
Academic Pr., 1947.

Treatment Services Bulletin, Ottawa (monthly).
V. 2 (1947).

Universities quarterly: London.
V. 1 (1946).

Year book of endocrinology, metabolism and nutrition.
V. 1 (1946).

(To Be Continued)